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PHYSICAL DIAGNOSIS.

PHYSICAL DIAGNOSIS

A GUIDE TO METHODS OF CLINICAL INVESTIGATION.

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TO THE

PHYSICIANS TO THE ROYAL INFIRMARY,

PAST AND PRESENT,

WITH WHOM WE HAVE BEEN ASSOCIATED

IN CLINICAL WORK.

PREFACE.

In the following work are embodied the results of several years' experience in teaching methods of clinical investigation. We have limited ourselves almost entirely to the consideration of physical examination, as this is the department of practical medicine in which students require most assistance.

While we have no desire to avoid our united responsibility for this work, it is right to indicate the portions for which we individually must be held to be chiefly responsible. The sections devoted to the Integumentary, Urinary, and Nervous Systems, together with the Examination of the Pulse, are by Dr. Gibson; the Circulatory, Respiratory, and Alimentary Systems are by Dr. Russell.

No attempt has been made, except in a few instances, where the original writings will be found to be of special use, to acknowledge the various sources from which we have derived our knowledge of physical diagnosis. To do so fully would be practically impossible, as it would not only entail an enumeration of most of the works dealing with the subject from the time of Laennec to the present, but also render it necessary to estimate the influence of intercourse with many minds, as teachers, pupils, and friends.

We have gratefully to acknowledge our indebtedness to Dr. M'Bride, Surgeon to the Ear and Throat Department of the Royal Infirmary, for the sections on the Ear, Throat, Pharynx, Naso-Pharynx, and Larynx; and to Dr. Mackay, Assistant Ophthalmic Surgeon to the Royal Infirmary, for those devoted to the Eye. These will, we believe, materially add to the value of the book.

We would also cordially express our obligations to Dr. Noël Paton for his kindness in revising the section dealing with the Urinary System, and for aiding us with many practical suggestions in regard to it.

We further wish to tender our thanks to Mr. William Keiller, F.R.C.S.Ed., for his efforts to realise our aim in the diagrams illustrating topographical anatomy and the physical conditions producing clinical phenomena.

EDINBURGH, *March* 1890.

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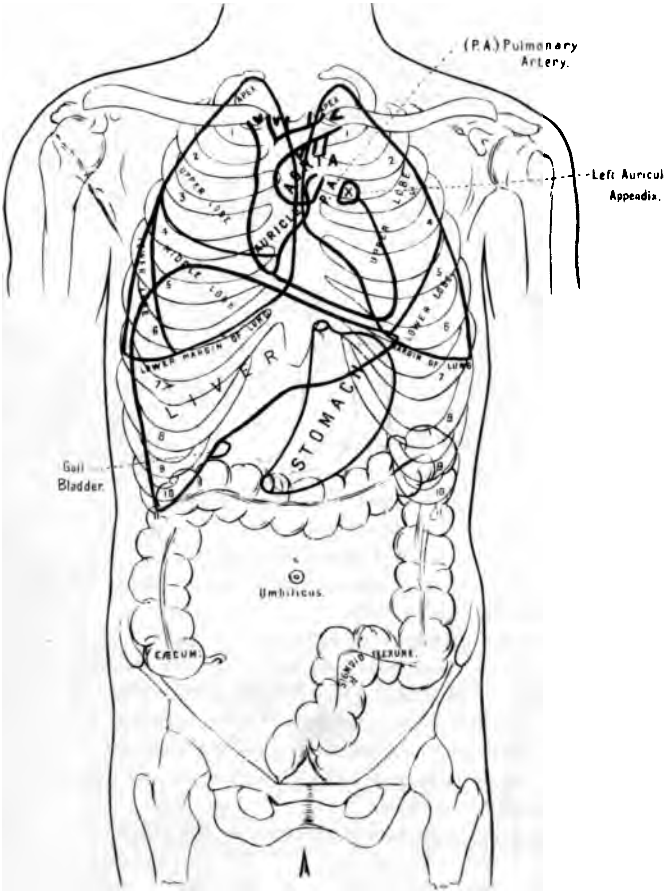
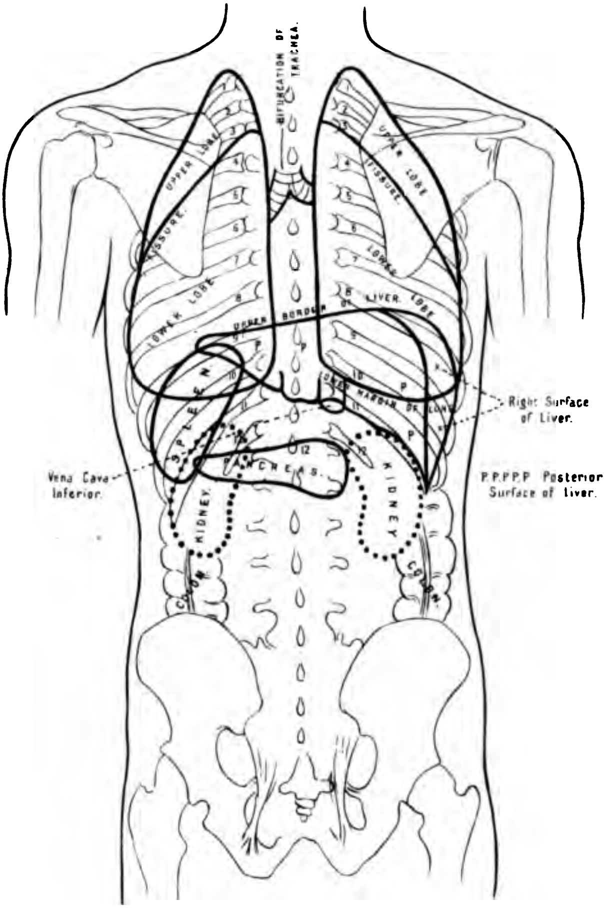


Frontispiece I.—Relations of Thoracic and Abdominal Viscera. Anterior view.



FRONTISPIECE II.—Relations of Thoracic and Abdominal Viscera. Posterior view.

PHYSICAL DIAGNOSIS.

CHAPTER I.

INTRODUCTORY AND EXPLANATORY.

In approaching the subject of diagnosis, we have to ask the question, How is a diagnosis made? and in reply, it may be said that it is a complex, not a simple process. The evidence on which it rests is derived from three main lines of inquiry. First, we ask the patient what he complains of; the complaints are the *Symptoms.* Second, we inquire as to the length of time the symptoms have lasted, and the order in which they appeared; these, and all facts bearing on the patient's constitution, mode of life, and family antecedents, constitute the *History.* Third, we examine the part or region to which the symptoms are referable, and at the same time examine the condition of the other systems, although no complaint may be made of them; this is *Physical Examination.* By means of the information derived from these three sources we then form a diagnosis or an opinion as to what the disease is under which the patient is labouring. In many cases it is quite impossible to form a correct diagnosis without considering the facts derived from all three sources; in other cases, however, it is possible to form an absolutely correct diagnosis from the physical examination alone, and an opinion formed in this way is what is strictly speaking physical diagnosis. In *all* cases, however, it is necessary to make a physical examination of the patient before forming a diagnosis, and it is with a view to help the student of clinical medicine in this important and difficult department that this book is written. In it we have in the main followed the lines which, in our experience as Tutors in Clinical Medicine in the Royal Infirmary of Edinburgh, have appeared to be most useful.

Physical Examination embraces and is confined to facts which are apparent to the senses of sight, touch, and hearing, and to these may be added smell.

The application of sight is known as *inspection*, touch as *palpation*, and hearing as *auscultation* or *percussion*, according to whether the sounds are produced by the organs themselves, or by tapping the different regions of the trunk.

It is of course necessary to be acquainted with what is to be seen, felt, and heard on examination of the different regions of the healthy body before the abnormal can be appreciated. It is necessary to be familiar with the normal shape of the abdomen, in order to describe the alterations which occur in disease; to know the character of the normal cardiac impulse, in order to appreciate deviations from it; and to have an intimate acquaintance with the normal sounds heard on auscultation, before studying the sounds present in disease.

Students ought, therefore, to avail themselves to the utmost of every opportunity of examining healthy individuals, and particularly healthy chests, both in hospital and dispensary practice; they will thus become familiar with the normal and the variations which occur within the limits of health.

Physical diagnosis, however, includes not only investigations by means of the senses; it also inquires into the physical causes or conditions which produce the various phenomena. For instance, on inspection, an abdomen is found to be distended; by the aid of percussion and palpation, and the application of an elementary knowledge of physics, it is decided whether it is distended with fluid in the peritoneal cavity or gas in the intestines. When this point is settled, we are in a position to state the various causes which may lead to the condition, but it is often impossible from this alone to form an opinion as to the cause in any particular case; this may only be possible after a careful consideration of the history and all the symptoms of the case. The possibility of forming a diagnosis from physical examination alone is distinctly limited, but no diagnosis ought ever to be attempted without making a physical examination. The symptoms and the clinical history of individual diseases will be found in the ordinary text-books of medicine, and they will not be dealt with at length in this volume.

CHAPTER II.

METHODS AND TERMS.

General Inspection.—By general inspection is meant the impression which the patient's first appearance makes on us, and also the observations we make during the time inquiry is being made as to his complaints. The trained eye learns much in this way as to the temperament, constitution, possible habits, and the system, which may be at fault; and the student has to train himself to be an observer of these. Mental notes are made as to the state of nutrition, the gait and carriage, the colour of the skin and hair, the apparent age, the expression of the face and eye, the voice, any apparent abnormalities in exposed parts, and so forth.

If the patient be seen in bed, similar observations have, so far as possible, to be made. In addition, however, are to be noted the decubitus, whether on the back or on the side, whether lying flat down or partially raised, whether the knees are drawn up or the legs extended, the look of exhaustion or of suffering, of consciousness or unconsciousness, of apathy or interest; the respiration, whether accelerated, or slow, or irregular, whether the nostrils move, or a look of suffering accompanies inspiratory efforts; if cough be present, whether it is "soft" or "hard," whether there is perspiration, whether the face is pale, flushed, or cyanotic. All these points will be noted almost automatically after a time if the observing faculties are carefully cultivated, and by that time their significance and value will be appreciated, but until then no reliable conclusions can be drawn from them, and meanwhile they, or any other abnormal appearances, ought simply to be recorded.

Local Inspection.—In addition to the foregoing, the inspection of the various regions, especially of the thorax and abdomen, is necessary. In this are included shape, degree of plumpness or emaciation, movements, and so forth. It may also be made to include *mensuration*, that is, the determination of the size of the part.

Palpation.—By means of the hand or hands placed on a part are noted its degree of resistance, its hardness or softness, whether fluctuation be present or not, the degree and kind of movement, the presence of abnormal sensations, and so forth. Under this head may be included *vocal fremitus*, a term which is confined to the examination of the lungs. It is the sensation which is felt by the hand when placed flat on the thorax while the patient speaks, or repeats the numeral " one, one, one," or any combination of numerals. The physical explanation of the phenomenon is that speaking sets up vibrations in the column of air in the trachea, that these vibrations are propagated into the air in the bronchi and air vesicles, and pass through the lung tissue and chest wall to the hand placed on it. Its intensity varies greatly in different individuals, and depends on factors which will be considered under palpation of the lung. For its production it is of course necessary that there should be no obstruction in the bronchi, as any such would arrest the passage of the vibrations.

Percussion.—This term is employed to denote the method of determining the physical condition of the viscera by the sound elicited on tapping the surface of the body. For a thorough understanding of the results obtained by this method of investigation it is necessary to consider the conditions under which sound is produced and conducted.

Sound consists of undulations produced in some body which has been caused to vibrate. These are transmitted through various media, by which they are conducted to the auditory apparatus. They thus reach the brain, where they are translated into different sounds. Sound-vibrations are longitudinal, that is to say, they occur in lines parallel to the direction in which they are conducted, and they are therefore to be regarded as waves of condensation and rarefaction.

There are two kinds of sounds—musical sounds, or *notes*, and non-musical sounds, or *noises*. The former consist of undulations possessing regular wave-lengths, and recurring rhythmically or periodically. The latter consist of vibrations without any definite length of wave.

Sounds differ in their intensity, pitch, and quality.

The *intensity* of a sound depends on the energy of the individual vibrations. A sound is louder, therefore, in proportion to the amount of force employed in producing these vibrations. The *pitch* of a sound depends on the number of vibrations in a given interval of time, and it becomes higher in pitch in direct proportion to the number of these vibrations. The *quality* of a sound depends on the special characters of the body whose vibrations are the cause of it.

Some of the sounds caused by percussion are noises,—those, for instance, produced by percussing the solid organs. The sounds thus elicited resemble those produced by striking such a body as a lump of clay. To such sounds the term *dull* is commonly applied. Other sounds again are notes, such as those produced by percussing the intestines when distended with gas. These sounds are as truly musical as the note obtained on striking a drum, and they are commonly termed *clear*. Most of these clear sounds, however, are obscured by being mixed up with the sounds produced at the same time by the solid organs, and the resulting sounds in such cases are not true musical notes.

As the use of the term *note* is apt to lead to confusion, the term *sound* will in the following pages be exclusively employed.

Percussion may be employed by tapping the surface of the body directly, either by means of the fingers, or by means of a specially constructed hammer which is called a plessor. This direct method is termed *immediate* percussion. The body may, on the other hand, be tapped indirectly through the medium of a finger laid flat on the surface, or of a specially constructed instrument called a pleximeter applied to the part. This method is known as *mediate* percussion, and is that most commonly employed.

Percussion is divided into *gentle* and *strong*, according as little or much strength is expended in its production; the general rule as to which ought to be used is that it ought to be—

Strong if the parietes be thick from fat or muscle, or if the organ whose limits are to be defined lies behind some other structure,—as, for instance, the left edge of the heart, which is covered by lung.

Gentle if the parietes be thin, or the organ lies superficially, or if there are structures of different physical characters from the one being investigated in close proximity to it; as, for example, intestine containing air behind the solid but thin anterior edge of the liver, or the solid liver behind the thin inferior edge of the right lung.

Auscultation.—By auscultation the sounds produced by some of the organs are heard. It is chiefly devoted to the auscultation of the sounds produced in the respiratory organs during respiration, and in the heart and large vessels as the blood is flowing through them. The sounds are due to vibrations set up in these organs and propagated through the chest and stethoscope to the ear.

It may be practised by putting the ear directly to the part, when it is called *immediate auscultation*; but it is more commonly performed through the medium of the stethoscope, and is then called *mediate.* In using this instrument one or two precautions are necessary. Grasp it between the two forefingers and the thumb, and place it perfectly flat on the part at which it is proposed to listen; adapt your own position and body so that the ear can be brought flat upon it without moving it. If this is not carefully attended to, the stethoscope rides on its edge, hurts the patient, and the conduction of the sound is interfered with.

The auscultator should assume as comfortable a position as possible, and he should avoid having his head hanging down. Further, he should be careful to lean with the hand not on the patient, but on the bed, and not to let the weight of the head rest on the stethoscope,—in fact, to lean as lightly as possible on it.

Vocal resonance may be included under this head. When the stethoscope is applied over the lungs, and the patient asked to speak, as described under vocal fremitus, a sound is found to be conducted through the stethoscope to the ear and it is to this conduction of the voice that the term is applied.

Combined percussion and auscultation, and other special methods, will be referred to when describing the physical examination of the various organs.

Succussion is the term applied to a splashing sound heard sometimes at a considerable distance, at other times only on auscultation, when the patient's body is shaken. It indicates the presence of fluid and air, and is of special importance when produced in the pleural cavity. It was first described by Hippocrates, and often has his name prefixed to it.

CHAPTER III.

TEMPERATURE.

THE temperature of the body is usually taken in the axilla; in young children it is better, as a rule, to take it in the groin. The ordinary clinical thermometer ought to be left in for five minutes. If the half-minute thermometer be used, it is usually placed in the mouth under the tongue, and it requires to be carefully cleansed after being used. Care has to be taken that the axilla is dry; for if wet or moist, the condensation of vapour on the glass prevents its heating, and the mercury does not rise.

The temperature of the axilla is normally one degree below that of the rectum. In some special cases it is necessary to take it there, as the temperature of internal parts may be elevated, while that of the surface may be even lowered. This occurs, for instance, in at least some cases of malignant scarlet fever, as well as in other malignant fevers.

A good deal of attention has been paid to surface temperature taken by means of thermometers with flat bulbs. This has been used for the investigation of intrathoracic and intracranial and other internal affections, but the results have not been sufficiently conclusive, so far, to lead to the general adoption of the method. The surface temperature is normally one or two degrees below that of the axilla.

Normally the temperature is slightly higher in the evening than in the morning, and this normal habit is usually adhered to in disease, although they may both be on a level much above normal, or the variations may greatly exceed the normal variation. Occasionally, however, in diseased conditions the order is reversed, and the temperature is higher in the morning than in the evening.

The temperature is a factor of much importance in disease, and as its height varies, it may be necessary not only to take it morning and evening, but at shorter intervals. As a rule, however, morning and evening observations are sufficient, and, save in exceptional circumstances, more frequent observations are not practicable.

In nearly all acute inflammatory diseases the temperature is elevated. In children, gastro-intestinal disturbances, due to indigestion, often produce a rise of temperature of several degrees. We have also seen this in old people, but it is rare in them. Certain individuals have a very unstable temperature equilibrium, a slight catarrh or "cold" being sufficient to raise their temperature, and to lead to considerable discomfort; while in others it requires a more serious condition to disturb the equilibrium.

The length of time the temperature is raised depends on the duration and the nature of the illness, and is often the most reliable index as to its progress. The onset of acute disease is heralded by, amongst other symptoms, a more or less sudden rise in temperature, its course by a more or less continuously elevated one, and its decline by a gradual or sudden return to normal.

Febricula is a short illness, lasting from twelve to forty-eight hours, usually inaugurated by a slight rigor, or a feeling of chilliness, and associated with a rise of temperature of several degrees. It frequently occurs in women a few days after delivery, and some women are subject to them at intervals, their precise cause not always being apparent. They are popularly known as "weeds." The chart Fig. 1 is from a case of this kind.

In illnesses of longer duration, the temperature curve presents some well-marked types.

1. First, there is the type of *continued or sustained fever*, followed by gradual decline and return to normal, this mode

of decline being known as *lysis*. It is typically seen in

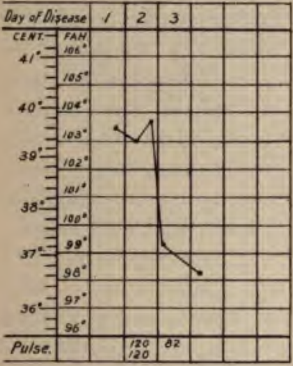


FIG 1.—Febricula, third day after delivery.

some cases of typhoid, but is not the type invariably shown by this fever. The annexed tracing is from such a case.

2. The chart Fig. 3 is also from a case of typhoid fever, in which the variations in each period of twenty-four hours is considerable. This type of temperature might be called the *regularly remittent*. It is often present in septicæmia, due to septic absorption from an abraded surface. In typhoid fever it is to be remembered that septic absorption from the seat of intestinal *ulceration is an element in the case.*

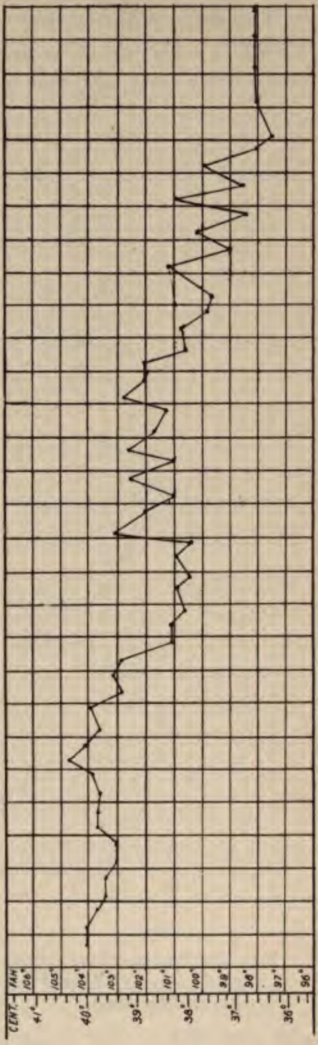


FIG. 2.—From a case of typhoid fever with sustained temperature—Recovery.

The temperature during the first days of the fever is not shown in either chart, as neither case came under observation sufficiently early, and this is the common experience. It is further to be noted that while the foregoing are types of temperature curve in typhoid, the duration of individual cases varies greatly, some being shorter than that represented in Fig. 3, while others are longer.

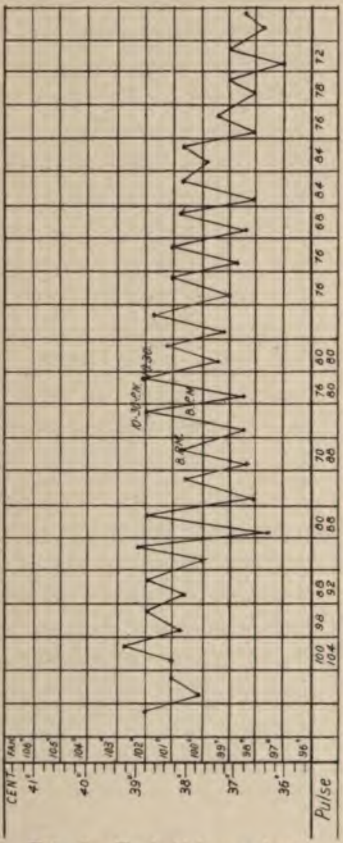


FIG. 3.—Typhoid fever, showing a regular diurnal remission—Recovery.

3. An *irregularly remittent* temperature is seen in phthisis and prolonged pyæmia, the variations of level being more extreme and much more irregular in them than in the preceding.

4. *Sustained* temperature, with a sudden fall to normal at the end of six to eight days, characterises acute croupous pneumonia. This sudden fall to normal is known as *crisis*, and is shown in Fig. 4.

While this sudden fall is characteristic of pneumonia, and indicates a favourable termination of the acute process, a similar fall, in typhoid fever for instance, would indicate a grave change for the worse, and probably a fatal issue from collapse, due to intestinal hæmorrhage or some equally serious condition.

5. *Intermittent* is the type of temperature present in malarial fevers. Here there is either a break of one day between the febrile paroxysms, when it is called quotidian, of two days, when it is called tertian, or of three days, when it is termed quartan. The above chart from Wunderlich represents these varieties.

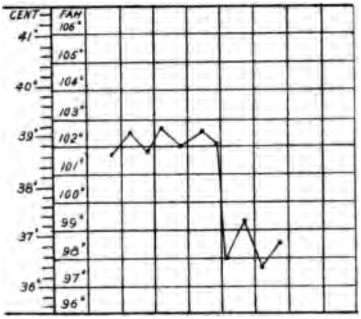
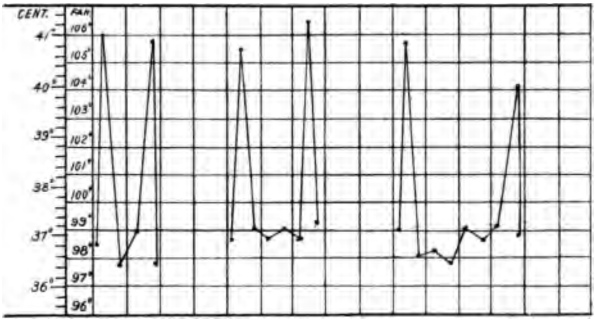


Fig. 4.—Acute pneumonia showing fall by crisis.



Quotidian. Tertian. Quartan.

Fig. 5.—Showing intermittent type of temperature.

6. *Relapsing* is the type of temperature represented in

Fig. 6. There is an interval of several days between the febrile periods; it is typically present in relapsing fever.

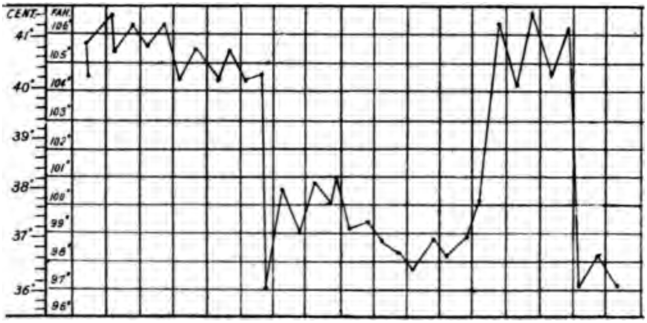


FIG. 6.—Showing relapsing type of temperature.—(Wunderlich.)

Hyperpyrexia is the term applied when the temperature rises above 105° F., and is accompanied by severe cerebral and general symptoms. It occurs in rheumatic, scarlet, typhoid, and other fevers.

When the temperature in acute or chronic disease suddenly rises above what it has reached for some days, it indicates the involvement of a hitherto unaffected part. We have seen this in rheumatic fever when the heart became affected, in gangrene of the lung with the onset of inflammation of the pleura, in purulent pleurisy with the onset of pericarditis, and so forth.

Relation of Temperature and Pulse rate.—The rapidity of the pulse usually increases *pari passu* with the rise of temperature. This is not invariably the case, however. In typhoid fever, for instance, the temperature may be 102° or more, while the pulse is only about 80. In tubercular meningitis in children a like want of proportion may be present in its earlier stages.

CHAPTER IV.

INTEGUMENTARY SYSTEM.

EXAMINATION OF THE SKIN.

CHANGES in the condition of the integument must be regarded from two points of view, as they may, on the one hand, be merely expressions of general conditions, while on the other they may be the result of strictly local disturbances. It is necessary in this way to regard the cutaneous and subcutaneous tissues as sharing in the state of the body at large, or as being the seat of more or less restricted lesions. These considerations lead to a division of the subject into the two heads of *general conditions* and *special affections* of the skin.

GENERAL CONDITIONS.

As very many disorders of the internal organs produce changes in the condition of the cutaneous and subcutaneous tissues, careful examination of these textures should in every case be made. The points to which attention should be directed are—

The colour of the skin.
The moisture of the skin.
The condition of the subcutaneous tissues.

In addition to these points the temperature of the skin is of the highest importance, but this is dealt with in the section devoted to the body temperature.

Changes in the Colour of the Skin.—The healthy tint of the European skin may undergo modifications in many directions.

Pallor of the Skin.—Paleness is due to deficiency of blood in the cutaneous capillaries, and this may be temporary or permanent.

Temporary pallor occurs from cardiac failure, as in emotional syncope and enfeebled conditions of the heart, such as fatty heart. It may also occur from constriction of the arterioles, as in such mental states as fear and pain, as well as under the influence of external conditions, as, for example, cold. Permanent pallor may be the result of a diminution in the number of the red corpuscles of the blood, or of a deficiency of hæmoglobin, as in chlorosis, anæmia, and leukæmia, as well as chronic Bright's disease and other affections. Or it may be caused by a reduction in the amount of the blood due directly to hæmorrhages, and indirectly to effusion of the serum or to deficient assimilation.

Pallor of the skin is, as a rule, attended by paleness of the mucous membranes, which are apt to be dry. The causes which induce it are apt to lead to dropsy, and when this is the case the pallor becomes increased.

Redness of the Skin.—Redness of the skin is due to hyperæmia. Temporary excitement, such as is frequently caused by an interview with a medical man, may cause dilation of the arterioles and rapid as well as powerful action of the heart, attended by flushing of the skin, but this rapidly subsides.

A more permanent redness, however, is one of the most frequent symptoms of acute diseases, as in the flush over the malar bones in acute pneumonia, in which it is of a dusky tint, and in hectic fever, where it is brighter in hue.

A dull diffused redness of the face, with arborescent markings of a purple or blue colour, caused by stasis in the venous radicles, and commonly termed *venous stigmata*, is common in chronic disease of the heart, involving the systemic veins, and is also very frequently a symptom of chronic alcoholism.

Blueness of the Skin, or Cyanosis, is the result of venous stasis, the dilated veins containing blood which is deficient in oxygen.

This condition is found in malformation, as well as disease, of the heart or pericardium.

In the malformations of the heart which permit the venous and arterial blood to mingle together, the blood which is supplied to the systemic arteries is partly venous.

Valvular lesions cause venous stasis by backward pressure from the left ventricle, or from inability on the part of the right cavities to forward the blood in the proper direction.

Muscular degeneration of the heart is similar in its results, and the pressure of pericardial effusion occasionally produces cyanosis.

Cyanosis may be due also to affections of the respiratory system. Temporary venous stasis is very frequently observed during fits of coughing, which prevent the proper circulation of the blood through the lungs. Permanent venous stasis may be caused by the prevention of the access of air by any narrowing of the larynx, trachea, or bronchi, *e.g.*, in croup. It may also be caused by lessening of the aërating surface of the lungs, and this may occur in such changes of the texture of the lung as emphysema, or in compression over a large area of the branches of the pulmonary artery, the result of external pressure from effusion into the pleura.

Local cyanosis is occasionally to be observed as the result of pressure on large veins.

The colour of the skin in cyanosis varies from a pale bluish tint to a dark blackish blue. When it is intense, the mucous membranes—as, for instance, the lips and gums—are also coloured, even the conjunctiva sharing in the venous stasis, and showing distinctly the distended veins. Pressure in this condition drives away the colour, which returns again immediately. It differs in this way from the colouring which is caused by the administration of nitrate of silver. The veins of the neck and arms are frequently distended, and may undulate with the movements caused by respiration and circulation. The skin is cool, and the nose and ears, as well as the feet and hands, are as a rule cold. There is a tendency to arborescent networks on the surface of the lower extremities, and sometimes intense cyanosis is accompanied by œdema of the feet and ankles.

Yellowness of the Skin.—A yellow tint of the skin, constituting icterus or jaundice, is present in many cases either of temporary or permanent interference with the functions of the liver, and sometimes from alterations in the constitution of the blood. As examples of temporary causes of jaundice, catarrh of the duodenum and gall-ducts may be mentioned, bodies within the ducts, such as gall-stones or parasites, and pressure on the ducts by external causes, such as accumulation of fæces in the bowel. Among the permanent causes, stricture and tumours of the gall-ducts, and pressure upon them by tumours of other viscera, deserve passing notice.

The other class, sometimes termed hæmogenic, includes the discoloration which sometimes follows the inhalation of anæsthetics, and also that which occurs in some acute general diseases, the absorption of some drugs, such as phosphorus and antimony, the virus of snake-bites, the poisons of many acute specific diseases, such as typhus, enteric, relapsing, malarial, yellow, and scarlet fevers, as well as epidemic jaundice.

Bronzedness of the Skin.—Changes in the colour of the skin may be caused also by disease of the supra-renal capsules. In this condition the skin assumes a bronze colour, more especially in those parts of the body which are darker than the rest.

Bronzing, but to a less extent, is found in many cases of pregnancy, and it may be a symptom of pelvic disease in women.

Greyness of the Skin.—The use of drugs may induce changes of colour; the prolonged use of nitrate of silver gives rise to a grey discoloration known as *argyria*, which must not be mistaken for cyanosis.

The Moisture of the Skin.—The amount of perspiration must be noted. It is greater at all times in the axillæ, perinæum, palms, and soles than over the rest of the body. The perspiration is increased or diminished by mental conditions. Its amount is habitually greater in those who inherit the rheumatic constitution, while in acute rheumatism it is profuse during the febrile stages of the disease. It constitutes the most important symptom of certain stages of disease, such as the sweating state of ague, the crises of continued fevers, the febrile attacks of tuberculosis, and the condition of collapse. Increased perspiration may occur locally in nervous diseases, being limited to the part supplied by the affected nerves. The perspiration is diminished in the early stages of fevers, and in such diseases as diabetes.

The perspiration has a characteristic odour in uræmia, and the sweat of the axillæ and other regions may have a fœtid smell from its decomposition.

In jaundice the perspiration may have a yellow tint from the bile pigments which it contains, and other colours have been observed in some rare cases.

In increased perspiration the skin may be dotted all over with very small vesicles corresponding to the sudoriferous gland. This condition will be more fully described under skin diseases.

Condition of the Subcutaneous Tissues.—The subcutaneous textures vary in their conditions as to nutrition. The amount of fat deposited in those tissues may be greatly increased or very deficient. An increase in its amount is often hereditary, but may be due to errors of diet and exercise. Fat may be deficient as a constitutional habit of body, or may be a symptom of wasting disease, and if it should have appeared suddenly, it points to the latter as the probable cause.

The subcutaneous tissues may be swollen from the presence of the serum of the blood, and this constitutes the condition known as œdema or anasarca.

The position of the œdema may point to its cause, for, as a rule, in cardiac anasarca, the swelling begins at the ankle, while in that of acute Bright's disease it appears primarily in the lower eyelid. The affected part may only appear to be a little larger than usual, but, on the other hand, the skin may seem to be greatly distended, and if this be so, it is pale, smooth, and shining. If the finger be pressed upon the swollen part, an indentation is produced, which fills up gradually when the finger is removed.

The causes of œdema may be cardiac, vascular, or renal.

Cardiac dropsy may be primary, and due to the retardation of the return of venous blood by regurgitation into the veins, or it may be secondary to chronic pulmonary diseases.

Vascular œdema may be due to disease of the coats of a vein, such as thrombosis following phlebitis, or it may be due to pressure on one of the venous trunks.

Renal dropsy, as has been mentioned above, makes its appearance usually first in the face, especially in the eyelids. It soon becomes general, and is accompanied by dropsy in the various serous cavities. The colour of the skin is pale and pasty looking.

Anasarca may be produced by deficiencies in the quality of the blood, which may be primary in anæmia, or occur in the course of wasting diseases.

The subcutaneous textures in the disease known as myxœdema are invaded by a gelatinous substance containing mucin. In this disease the whole surface of the body is swollen. The face becomes large, swollen, and expressionless, the hands blunt and clumsy, and along with these conditions there is hebetude of the nervous system. In myxœdema there is no indentation on pressure with the finger, and it affects every part of the body alike.

The subcutaneous tissues may be swollen from the presence of air. This is known as subcutaneous emphysema. It may be caused by disease of, or accident to, the larynx or trachea, but it is much more commonly caused by a rupture of the tissues of the lung, which allows the air to escape from the air vesicles or bronchioles into the connective tissues, by which means it reaches the root of the lung, and thence passes by the mediastinum to the neck.

In this condition the skin does not pit on pressure, or does so very slightly, and the finger feels a distinct sensation of crackling when it is passed over it.

SPECIAL AFFECTIONS.

In studying the appearance of any skin disease, as much of the surface of the body should be seen as may be possible or expedient, and in the course of the examination attention should be systematically directed to the locality of the disease, the outline of the affected regions, the type of the eruption, the nature of the lesion, and the structure invaded.

Distribution.—The situation of the disease is a most useful guide in diagnosis. Certain affections are confined to particular spots—sycosis, for example, to the hairy parts of the face. Others have areas grouped anatomically, as in the case of eczema and psoriasis, the former of which exhibits a preference for the palms, soles, and flexor surfaces of the extremities, while the latter attacks the elbows, knees, and extensor aspects. Others, such as pityriasis rubra, are scattered indifferently over the body. Others again invade the whole surface, as may be seen in the cutaneous phenomena of the exanthemata.

These remarks must not be accepted in the sense of absolute rules, for the points laid down are not of universal application. Diseases which are at first limited to definite regions have a tendency to spread from these areas; scabies, to take a common example, although at the onset confined to the hands, and more especially to the webs of the fingers, invariably extends upwards along the arms, and frequently attacks those parts of the body which are most likely to come in contact with them. On the other hand, those diseases which have been mentioned as involving the entire surface of the body are commonly found to be more fully developed in certain regions; in measles, for instance, the rash, which is diffused over the whole body, is always much more distinct on the face, and more especially on the forehead. With such reservations the statements made regarding the distribution of the disease are nevertheless of very general application.

Conformation.—Where the disease is found in separate areas the outline of the affected spots may afford useful information, especially in cases of a parasitic or syphilitic nature. In ringworm, for example, the original shape of the patches is circular or oval, and dermato-syphilis has a tendency to assume circular and gyrate outlines.

Type.—According to a time-honoured arrangement, the various disorders of the skin are grouped together in the two classes of primary and secondary diseases. To the former belong *maculæ* or stains, *papulæ* or pimples, *pomphi* or wheals, *vesiculæ* or blebs, *bullæ* or blisters, *pustulæ* or pocks, and *tuberculæ* or growths. In the latter division are placed *squamæ* or scales, *excoriationes* or abrasions, *ulcerationes* or sores, *rhagades* or cracks, *crustæ* or scabs, and *cicatrices* or scars. These terms are very useful when employed simply as definitions of certain appearances, but if used as a basis for classification they are apt to lead to confusion, since very different pathological processes may be brought together by means of them.

In every case it is necessary to note the type of the elementary lesion, and to distinguish the essential from the accidental appearances; in pediculosis, for instance, the hæmorrhagic points, which constitute the primary or essential lesion, must not be confused with the excoriations and crusts, which are only secondary or accidental phenomena.

Seat.—In by far the larger number of the changes which it undergoes the skin is affected throughout all its layers. There are, nevertheless, a few diseases which are confined to one or other of its structural divisions, and although it may not be easy in the fully developed condition to determine the elements involved in any morbid process, there is no difficulty at the outset in assigning an affection to its true anatomical position.

The *epidermis* is seldom the only seat of cutaneous diseases, but it necessarily suffers in the course of most of them. Its horny layer is specially affected in psoriasis and pityriasis, as well as in ichthyosis and xeroderma, while deposits of pigment and the formation of vesicles take place in the mucous layer.

The *corium* is the starting-point of the great majority of skin diseases, the papillary layer being the primary seat of all diseases dependent on changes in the circulation of the skin, and the areolar layer becoming involved during their course.

The *appendages* of the skin are the special seats of certain affections, and attention must be bestowed on the nails, the hair, the hair follicles, the sebaceous glands, and the sweat glands.

As mentioned above, the different changes will be considered in the light of their pathology, and in doing this their special anatomical seats will be pointed out.

Nature.—The character of the morbid process must in every case be recognised. One or two skin affections are purely subjective, but these do not fall to be considered in a work on physical diagnosis. The processes giving rise to the appearances which have to be observed are hyperæmias, exudations, œdemas, hæmorrhages, hypertrophies, atrophies, new formations, and parasitic invasions. In approaching these different disturbances it is better to consider the skin apart from its appendages, and to deal with the latter separately.

Method of examination.—The patient should be examined in a good light, and as much of the surface of the body should be seen as is possible, either at once or in successive areas. Partial examination is apt to lead to error from the multiform character of certain diseases. When scales or crusts are present they must be removed in order to expose the subjacent structures. In the course of inspection the sense of touch must be called to the aid of sight. The feeling of resistance helps the observer to estimate the condition of the skin and subcutaneous tissues, and to ascertain the extent of any changes in the cutaneous circulation. When redness of the surface is due to simple congestion, pressure completely removes the bright flush, and induces a temporary pallor; when the dilatation of the vessels is accompanied by serous infiltration, the removal of the ruddy tint is not so complete, and when the vascular changes have resulted in cellular proliferation, the colour is but little affected by pressure.

Pressure also enables the observer to distinguish between spots of hyperæmia on the one hand, and of hæmorrhage or pigmentation on the other. In the former the colour at once disappears, while it is unaffected in the latter.

In any affection of the hair it is necessary to pull out specimens of it for microscopic examination, and if the glandular apparatus be involved, a lens should be employed to ascertain what changes have occurred. Where there is any suspicion of a parasitic origin specimens must be examined under the microscope, for the purpose of detecting the nature of the parasite.

AFFECTIONS OF THE SKIN PROPER.

Hyperæmia.—Hyperæmia of the papillary layer causes a slight swelling of the skin, with a smooth surface and a bright flush which fades on pressure. The flush, termed a *macule*, has a pink colour in erythema, a rosy hue in roseola, and a lurid tint in the mottling of typhus. Diffuse hyperæmia occurs as part of the lesion in several skin diseases, which will be mentioned later in connection with their more characteristic appearances.

Hyperæmia of distinct papillæ gives rise to the formation of *papules*, which are small rounded elevations. Flesh-coloured papules arranged in groups are found in prurigo, and dull red papules, also aggregated together, are characteristic of lichen. Papules occur in the course of several acute general diseases. In typhus they are rosy and have a tendency to become petechial; in enteric fever they are pink and appear in successive crops; in scarlatina they are minute and scarlet, usually coalescing to form a uniformly distributed rash: in measles they are dull red and somewhat crescentic in outline; and in rötheln dull red, but paler than in measles, grouped in clusters over the body, but uniformly distributed over the face. Papules form the initial stage of eczema and other skin diseases to be noticed presently, and they are also found in the early period of small-pox, cow-pox, and chicken-pox.

Hyperæmia of the vascular layer, along with exudation around the papillæ, gives origin to *pomphi*, which are rounded elevations with a pale centre and a red halo, highly characteristic of urticaria or nettle-rash. Firm pressure causes the disappearance of the coloured zone.

Accumulations of serum, consequent upon hyperæmia, and having their seat in the rete Malpighii, or between it and the horny layer, constitute *vesiculæ* and *bullæ*, which are only distinguished from each other by their difference in size. Vesicles, which should not, strictly speaking, exceed a pea in size, are found in hydroa, where each bleb is situated on a distinct red base, and herpes, in which they are at first separate and surrounded by red zones, but afterwards become closely grouped together, and frequently coalesce. It is to be noticed that in herpes zoster the vesicles are developed along the course of distribution of nerve trunks. Vesicles occasionally develop at the summit of the papules in lichen, and form the second stage of eczema; they not infrequently attend erysipelas, and are highly suggestive of scabies when localised about the hands, and more especially the webs of the fingers, forming as they do the almost invariable sequel to the irritation of the itch insect. They also constitute a highly characteristic but transitory phase in the eruptions of variola, vaccinia, and varicella; in these diseases the vesicles are preceded by ruddy papules.

Bullæ are, as a rule, formed by the union of several vesicles, and are to be seen mainly in two diseases. In pemphigus, sometimes called pompholyx, a number of vesicles appear, which rapidly blend together in one large blister, whose contents are at first clear and afterwards milky from suppuration. The bulla subsequently bursts, and a crust is formed with a tendency to ulceration into the corium, but in cases that recover no scar is left. Rupia follows a somewhat similar course, but is distinguished by the severity of the ulceration beneath the horn-like crusts, and by the high degree of pigmentation of the scar which always results from the ulceration.

Pustulæ are small, superficial abscesses, seated in the mucous layer, or between it and the papillary layer, most commonly evolved from vesicles, the contents of which become purulent, or resulting from inflammation of papillæ. Pustules are present in one form of eczema, and in ecthyma they attain a large size. Superficial ulceration is a frequent result in these diseases, and when this occurs scars are left behind. Pustules form the third stage of the skin eruption in variola, vaccinia, and varicella, and leave in these diseases more or less cicatricial tissue behind.

Deeper inflammations of the skin are present in erysipelas, boils, and carbuncles. Erysipelas is characterised by its rosy colour, which fades on pressure, by considerable swelling, which may pit on forcible application of the finger, and by smoothness of the surface. Frequently vesicles or bullæ appear on the surface from serous accumulations, or pustules from superficial suppuration. Boils usually have their starting-point in a hair follicle, sebaceous gland, or sweat gland, and after passing through a papular stage become pustular, with a slough or core in the centre. Boils leave scars behind on healing. Carbuncles commence by an inflammation of the subcutaneous connective tissues, rapidly sloughing; and covered at first by the tense dusky red skin which is smooth and hard. Afterwards the skin becomes penetrated by the products of the inflammation, and vesicles or pustules are seen. After healing of the cavity left by the separation of the slough, more or less scarring remains.

Amongst localised inflammations involving deeper structures must be mentioned elephantiasis arabum. This affection begins by an inflammation restricted to one part, such as a leg or the scrotum, with lymphatic irritation, both vessels and glands being involved, and œdema of the part. It subsides, leaving a certain amount of swelling, but recurrrent attacks gradually lead to hypertrophy of the affected part, with a discoloured, eczematous, or even ichthyotic appearance of the skin.

During all inflammatory affections of the skin there is a tendency to the formation of *squamæ* or scales from the proliferation and desquamation of the cuticle. *Fissures* result from cracking of the epithelium, *excoriations* from its removal, and *ulcers* from extension of the morbid process to the deeper layers. *Crusts* are formed by collections of serum or pus with epithelial scales, and *scars* remain as a record of destructive inflammation.

Hæmorrhages into the skin may be caused by internal or external causes affecting the papillary layer. They occur in the papules of typhus fever, and sometimes in scarlet fever, measles, and small-pox, as a result of the inflammatory action; in scorbutus and purpura in consequence of the state of the blood-vessels; in cardiac and renal diseases from disturbances of the circulation; and they are induced by *external* violence in blows or falls, as well as by the biting or sucking of such insects as the bug (*cimex*), flea (*pulex*), louse (*pediculus*), caterpillar (*bombyx*), mite (*leptus*), tick (*ixodes*), or other parasite of the class. Hæmorrhages are classified according to their size and form. They are termed petechiæ when they are small points, vibices when longer in shape, and ecchymoses when of larger size. Their appearance does not change when pressure is applied, and their colour undergoes changes with lapse of time, passing from pink through crimson and purple to blue, and fading to green and yellow before resuming the usual tint of the skin.

The epithelium of the skin undergoes *hypertrophy* as the result of increased nutrition consequent upon chronic hyperæmia of the papillary layer in pityriasis rubra, lichen ruber, and psoriasis. In these diseases scales are freely thrown off, and are respectively whitish, greyish, and silvery in appearance. In xeroderma and ichthyosis the cuticle becomes hypertrophied in consequence of deficient secretions, without any hyperæmia. In these affections the scales are adherent and give a harsh dry and wrinkled appearance to the skin. Hypertrophy of limited areas from the effects of external pressure gives rise to callosities and corns, and when the papillary layer is also increased, warts or horns are developed.

Hypertrophy of the connective tissue of the skin gives fibroma, characterised by scattered tubercles over the surface of the body; cheloid, which is nodular, only one nodule as a rule being present, slightly raised above the level of the skin with branching extensions, and hard to touch; scleroderma, indurated and waxy in appearance and harsh to the touch; and xanthelasma, where the invading connective tissue is affected by fatty degeneration, giving a yellow colour to the affected patches, which may be flat or nodular, and are always soft to the touch.

An increase in the pigment of the rete mucosum gives rise to lentigo or freckles, occurring as little points or patches of yellow or brown colour, and to chloasma, sometimes called ephelis, which is found in larger areas of the same colour as freckles. These changes are for the most part confined to the face. In Addison's disease there is a great increase of pigment of a bronze tint, sometimes affecting the whole surface of the body, at other times involving definite areas, particularly the face, neck, axillæ, groins, genitals, and mammary areolæ. When in patches these shade off into the surrounding skin. Melasma is the term used for the discoloration caused by light and heat, which is apparently identical with that which results from the application of some irritant, such as a blister, to the skin. These pigmentary deposits must be distinguished from the discoloration in pityriasis versicolor, due to adherent scales caused by a parasite, and from that of icterus and argyria, which have been referred to. They are all alike in one point, that no change of colour takes place on pressure.

Vascular hypertrophy gives rise to nævi, composed of dilated and tortuous blood-vessels, bound together by fibrous tissue. Sometimes these are merely stains, not raised above the level of the skin, sometimes, on the other hand, they form distinct tumours. Their colour is usually crimson or purple.

Hypertrophy of the whole skin, with œdema of the subcutaneous tissues, occurs in elephantiasis arabum, which has been mentioned under the heading of deeper inflammations of the skin.

Atrophy.—A diminution of the pigmentation occurs as a general condition in albinism, and in the form of patches, which are surrounded by zones where the pigment is excessive, in the affection called vitiligo or leucoderma.

Among *new formations* invading the skin, syphilis, lupus, and leprosy are composed of granulation tissue, while the various varieties of epithelioma are formed by tissue of carcinomatous type.

In tubercular syphilides, as they are called, there is a development of small, smooth rounded tumours, usually of a dusky or tawny colour, sometimes irregularly scattered over the surface of the body, at other times arranged in circles or ellipses. These tubercles, as a rule, terminate without ulceration by subcutaneous absorption, and leave small depressed cicatrices behind.

Lupus begins with the appearance of small, rounded, dull-red swellings, arranged in a patch, which enlarges by the formation of additional tubercles. These tubercles may terminate in spontaneous cicatrisation, in superficial suppuration, or in deep ulceration.

Leprosy occurs in two forms, the tuberculated and non-tuberculated or anæsthetic. Tubercular leprosy begins by an elevated spot of ruddy tint, more darkly coloured in the centre and abruptly marked off from surrounding skin. This fades, and is succeeded by the development of tubercles, either in the affected spot or in the previously healthy skin. During the presence of the coloured eruption the areas involved are distinctly hyperæsthetic, and when the tubercles appear the affected regions are as markedly anæsthetic. The tubercular eruption has a great tendency to ulcerate, and during this change there is a foul smell. Tubercular leprosy is most common on the face, mammæ, genitals, and extremities. Anæsthetic leprosy makes itself known by localised attacks of pain and tenderness, especially confined to the arm and leg, and particularly along the course of the ulnar nerve, caused by a deposit of new tissue involving the nerves. After this has for some time been present, pigmented or blanched spots appear, especially on the back, shoulders, arms, nates, and thighs, attended by anæsthesia, but without any tendency to spontaneous ulceration. As the sensibility, however, is greatly impaired, bruises, cuts, and burns are frequently met with, and the wounds take on an unhealthy action, ending in ulceration and gangrene.

Epithelioma assumes the most varied forms, but ulceration is the most characteristic appearance which it presents. It usually begins as a small tubercle, with spreading induration and subsequent ulceration, but there may be simply an ulcer with scarcely apparent hardness around it. In the former case the disease tends to spread rapidly; in the latter it is much more gradual in its progress.

AFFECTIONS OF THE APPENDAGES OF THE SKIN.

The nails undergo changes from certain circulatory disorders, and it has already been said that in the cyanosis resulting from chronic circulatory and respiratory affections there is a tendency to clubbing of the fingers and arching of nails. In severe illnesses affecting the general nutrition the growth of the nails becomes impaired, and they show, after recovery has taken place, transverse depressions corresponding in their distance from the lunula to the length of time that has elapsed from the date of the illness. In addition to this impairment of growth from general causes, the nails may atrophy or hypertrophy irrespective of the state of the system as a whole. The nails undergo chronic inflammatory processes in pityriasis, lichen, and psoriasis, and the matrix is the seat of acute inflammation in onychia. This affection may be simple or syphilitic in its nature, and it varies in degree from a gentle subacute inflammation leading to indolent ulceration and loss of the nail, to severe erysipelatous inflammation, often going on to necrosis.

The hair may undergo changes in its amount, in its texture, or in the quantity of pigment which it contains.

An abnormal growth of hair in regions which usually have but little is known as hirsuties or hypertrichosis. The opposite condition, a deficiency of hair in those places usually covered, is known as baldness, alopecia, or atrichosis.

Changes in the texture of the hair lead to a fragile condition termed trichorexis, in which the hair breaks across, leaving a ragged end.

Diminished development of pigment leads to grey or white hair, and the condition is termed canities.

The parasitic diseases of the hair will be described afterwards, along with the other parasitic skin diseases.

The sebaceous glands are liable to be affected by certain conditions, which may best be grouped in three classes :—

excess, deficiency, or alteration of the secretion, retention of the secretion, and hyperæmia of the gland.

An excessive secretion of sebaceous matter constitutes the condition termed seborrhœa, or, more correctly, steatorrhœa. This occurs in two forms, one in which the secretion is oily, and the other in which it dries up in the form of crusts and scales.

A diminished secretion, or asteatodes, is often found in wasting, especially connected with such diseases as diabetes, and it is one of the symptoms of xeroderma and ichthyosis, which have already been mentioned under their proper head of hypertrophy of the epithelium.

An altered appearance of the secretion is on rare occasions met with, and receives the term allosteatodes. The most frequent change is one of colour, and the two tints which have been seen most commonly are yellow and black.

Retention of the sebaceous secretion *in the follicles* is seen in comedones, small papules with a central pit of dark colour, due to accumulated dirt. It is also pent-up *within the glands* in milium, formerly termed strophulus albidus. This affection is caused by obliteration of the ducts, and the appearance is that of small rounded papules of whitish colour. The *entire gland* is distended by retained and altered secretion in molluscum, where each lesion consists of a soft umbilicated tumour of a circular or oval outline, usually with a small opening into the interior, through which the yellow contents may be seen and squeezed out. Lastly, in sebaceous cysts, often growing to a considerable size, the secretion, usually altered and smelling foully, is retained within a strong lining membrane. Such cysts appear to be caused by blocking of the ducts.

Hyperæmic changes consecutive to retention of the secretion take place in the sebaceous glands, giving rise to two well-marked affections.

In lichen scrofulosorum there are groups of yellowish or brownish red papules seated at the opening of the follicles, and caused by exudation into the tissues around the glands. These papules frequently become purulent.

Acne is characterised by the presence of distinct papules appearing at the seat of comedones, which are the exciting cause of the hyperæmia, with a great tendency sometimes to induration, and at other times to pustulation. When the pustulation is severe, pits of cicatricial tissue are left behind, as in small-pox.

Like the sebaceous, *the sudoriparous glands* are apt to be the seat of diseased action. This shows itself by changes in the amount and nature of the perspiration, or by retention of the secretion.

The perspiration may be excessive in amount. Such a state is termed hyperidrosis, and it is usually the result of a constitutional condition, the effect of some nervous influence, or the accompaniment of the later stages of the febrile condition. When it is at once increased in amount, and at the same time retained, the condition of dysidrosis, to be mentioned below, is caused.

The sweat is diminished in some general diseases, as diabetes and the early stages of the febrile state, as well as in such skin diseases as xeroderma and ichthyosis. A lessened amount of the secretion is termed anidrosis.

Changes in the nature of the perspiration are often grouped together under the generic term paridrosis. When the perspiration putrefies readily there is an intensely disagreeable odour, and the condition is called bromidrosis or osmidrosis. It is at times highly charged with effete nitrogenous substances, giving the characteristic smell of urine. The condition has been termed uridrosis, and is common in chronic renal diseases. The colour of the sweat has been observed to undergo changes, and a term chromidrosis has been coined to meet this exigency. Coloured perspiration has been described by many writers, and appears to be caused by the presence of pigment derived from indican or from blood.

Retention of the perspiration gives rise to the formation of little vesicles containing clear fluid. The common cause of these vesicles is inactivity of the gland, as in the early stage of a fever, allowing the collection of epithelial cells over the opening of the duct, followed by renewed activity, as in the crisis of a fever. When the vesicles—which, as already said, are in the horny layer—contain clear fluid, the condition is that of sudamina, and when the contents are milky the term miliaria is employed.

When the perspiration is increased in quantity, and at the same time is retained by epithelial cells, painful and tender vesicles appear, with a great tendency to coalesce and form bullæ, terminating in exfoliation and excoriation. This condition is known as dysidrosis.

PARASITIC SKIN DISEASES.

Certain appearances, caused by the attacks of insects, have already been noticed amongst the hæmorrhagic affections of the skin, and require no further comment in this place. There are, however, some extremely definite disorders due to animal and vegetable parasites, which must be fully described.

Diseases due to Animal Parasites.—Pediculosis or phtheiriasis is the term employed to denote the ill-defined effects arising from the presence of one or other of the species of *Pediculus*, or the louse. The *Pediculus corporis* lives in the clothes, but feeds on the blood of the person subject to its attacks. The insect, whose body is oval in outline, is pale grey in colour, and is provided with a proboscis, which it inserts into the cutaneous follicles in order to suck the blood of the victim. The characteristic lesion is a pale papule, with a point of hæmorrhage in its centre. This is intensely itchy, and leads to scratching, which causes such secondary effects as hyperæmia and excoriations, followed by pustules and crusts. The *Pediculus capitis* lives in the hair of the scalp. It is a smaller insect than the *Pediculus corporis*; it has a triangular head, and dark markings on the sides of its body. It deposits its eggs on the hairs, each egg being attached to a hair by a short stalk and furnished with a lid. By sucking the follicles of the scalp they cause considerable itching, and the scratching which follows induces degrees of irritation which may even reach suppuration and swelling of the glands of the back of the head. The *Pediculus pubis* is found amongst the hair of the pubes, to which it firmly clings. It is still smaller than the species last described, and has a very irregular form. By its attacks it causes various lesions on account of the scratching induced by the irritation set up.

Scabies is caused by the *Acarus scabiei*, which burrows in the cutaneous tissues. This arachnid is oval in shape, and is provided with strong jaws and four pairs of legs. The male animal is smaller than the female, and has a sucker on each of the hind legs. The upper surface in both has a number of spinous processes, which are directed posteriorly. The eggs are oval in outline, and have a double line as seen under the microscope. The young acarus presents different appearances, according to its degree of development. The female burrows in the epidermis, forming elongated tunnels or cuniculi, in which she deposits the eggs. The male does not burrow so deeply. The cuniculus is a white or grey line, unless it is rendered darker by dirt, and is often obscured by a papule or vesicle caused by the irritation set up by the parasite. The most common seats of these burrows are the webs of the fingers, the inside of the wrists, the front of the thighs, and the front of the abdomen.

As a consequence of the itching, the patient scratches the affected parts and causes a great variety of lesions, which for the most part are apt to simulate eczema.

Diseases due to Vegetable Parasites.—The vegetable parasites which cause diseases of the skin consist in every case of a mycelium with spores, and it is of interest to note that they may be artificially cultivated in suitable media.

Tinea favosa or favus is caused by the presence of a parasite named *Achorion Schönleinii*, and occurs in small patches. At first these patches are somewhat red in colour, and the hair upon them looks dull and dry. They afterwards become scaly, assume a yellow colour, and, from being fixed in the centre, have a cupped appearance. When the yellow crusts are removed, the surface below is greasy, and may have a mouse-like smell

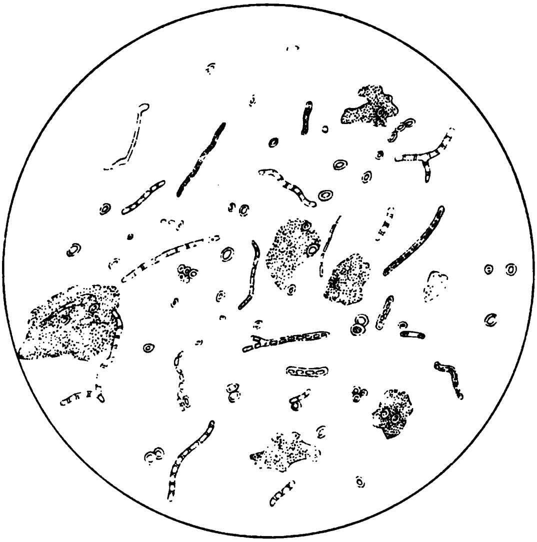


FIG. 7.—*Achorion Schönleinii.*

The crusts, on microscopic examination, are found to consist of the mycelium and spores, with epithelial cells, pus cells, and granular débris. In order to obtain a clear view of the parasitic growth, a piece of one of the crusts is to be soaked in water containing some liquor potassæ before examination. It will be found, as in Fig. 7, to consist of long branching filaments of elongated bodies united to form rows, and of small spores.

Tinea tricophytina is the result of the growth of the *Tricophyton tonsurans*, and, according to the situation of the disease and its corresponding effects, it is subdivided into three varieties.

Tinea tonsurans, or ringworm of the scalp, affects the hairy portions of the head. It makes its appearance in the form of circular or oval patches, varying in colour with the complexion of the patient, from straw to lavender. These are sometimes, but not often, surrounded by a zone of small vesicles. After a little time the patches become branny from the development of fine scales of epithelium, and stubbly because of the short, rough, broken hairs which project from them. It leads to areas of more or less complete baldness. If there is any difficulty in the diagnosis at an early period of the disease, Duckworth's chloroform test may be employed. This consists in the application of chloroform either to the growing hair, or to hair which has been pulled out; the chloroform is allowed to evaporate, and the hair when dry is found to have assumed a white colour if diseased.

Tinea barbæ, or ringworm of the beard, makes its appearance in the form of circular spots of a red colour, which afterwards become scaly and have a tendency to induration. The hair becomes dry and dull, and liable to break up or fall out, while the follicles frequently give rise to the formation of pus, leading to crusts.

Tinea circinata, or ringworm of the body, usually begins as a small pink or rose spot of circular shape, which may be vesicular round its margin. The colour fades at the centre, and the patch begins at this point to assume a scaly appearance, while it steadily extends in all directions. At times the lesion may be more severe than this, and the patch may become thick and hard from serous infiltration, becoming not at all unlike chronic eczema.

In the two first-named varieties of ringworm the hairs should be carefully examined under the microscope, and this should also be done with the scales in ringworm of the body.

The hairs and scales should be soaked for a short time in diluted liquor potassæ before examination, in order to render the recognition of the parasite possible. It appears as mycelium and spores, the former being in the form of long slender filaments which are jointed; the latter are small and round. This is shown in Fig. 8.

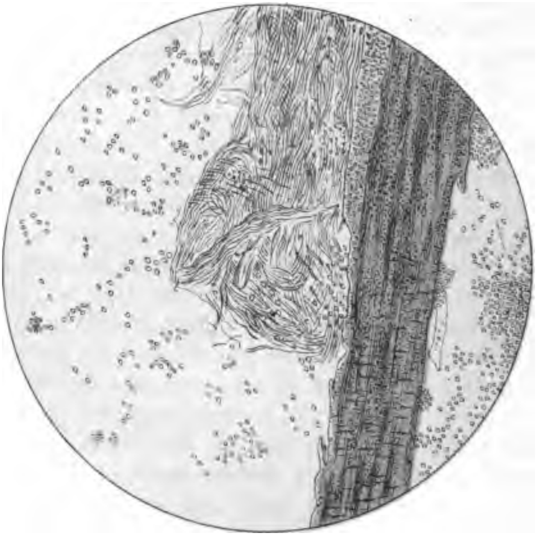


FIG. 8.—*Tricophyton tonsurans.*

Tinea versicolor is caused by the growth of a parasite called *Microsporon furfur* in the skin. It occurs in the form of raised patches of irregular shape, which vary in colour from a pale fawn to a dark brown, and are rough from the presence of branny scales, easily removed. These patches tend to increase in all directions, and by coalescing form large areas of discoloured skin. It is rarely found on any of the exposed parts of the person, the most common seats being the front and back of the body. By being scaly and elevated above the general surface of the skin it can, as a rule, be easily distinguished from chloasma, but in case of doubt the microscope should be used.

When a few of the scales are soaked in liquor potassæ and examined under the microscope the parasite may at once be detected. It consists, as in Fig. 9, of a mycelium, the filaments of which interlace to form a network, and round spores which are seen in groups within its meshes.

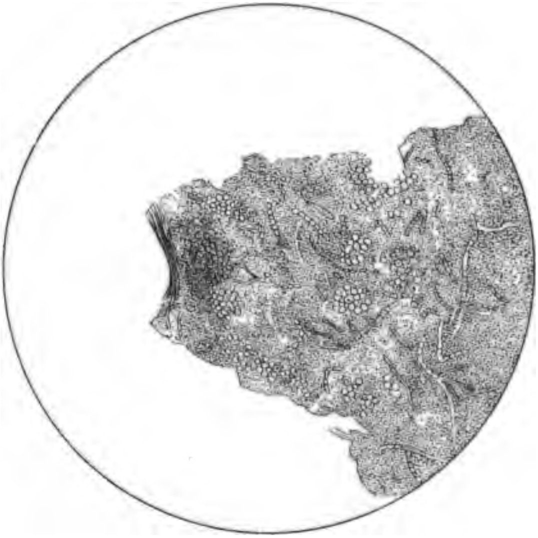


FIG. 9.—*Microsporon furfur.*

SYPHILITIC SKIN DISEASES.

A word may be said with regard to the characters of those skin diseases which are the result of syphilis. It cannot be too strongly urged that this affection may be the cause of almost every type of eruption, and it may be laid down as a general rule that there is usually an entire absence of all subjective symptoms.

Too much has perhaps been made of the colour of the skin affections. These are commonly described as being of a copper colour, but this is to be taken with some reservation. In the early rash of syphilis the colour of the eruption is pink or rosy, but the longer the rash remains the deeper becomes the tint, various shades of a ruddy brown hue, including a copper colour, being reached, and a pigmented area being frequently left after the disappearance of the rash. This pigmentation is a very frequent sequel to all kinds of syphilitic skin diseases; even in rupia the bright white scar left after the ulceration has healed is surrounded by a dusky zone.

The form of the disease is to be noted; it is very commonly circular or crescentic, or it may be the result of the union of several patches of such shapes, giving rise to more complicated outlines.

The distribution is very commonly symmetrical in the early manifestations of the disease, but this tendency to symmetry is not nearly so often to be seen in the later rashes.

The situation of the rash in early syphilis is usually on the front of the body or the flexor aspect of the limbs. Here again the later manifestations of the disease are not so regular in their site.

One of the most characteristic points in regard to cutaneous syphilides is the great tendency to the co-existence of several types of disease side by side. If several distinct kinds of skin disease are present at one and the same time, the fact would strongly point to the probability that they are caused by syphilis. In all such cases the whole body should if possible be thoroughly examined.

When syphilis is of long standing the skin assumes a pallid and muddy tint, which is extremely suggestive to the skilled eye.

CHAPTER V.

CIRCULATORY SYSTEM.

ANATOMICAL RELATIONS OF THE HEART AND GREAT BLOOD-VESSELS.

LIMITS AND RELATIONS OF THE HEART.

FOR purposes of physical examination it is convenient to regard the heart as presenting a base, an apex, and three sides. The *base*, as seen from the front, is formed from right to left by the superior limit of the right auricle and its appendix, and the junction of the pulmonary artery with the conus arteriosus of the right ventricle. It extends approximately from the level of the third left costal cartilage, a short distance from the edge of the sternum, to the corresponding rib on the right side, or to the interspace above it—that is, the second interspace—and to about an inch to the right of the sternum. The *apex* is under the fifth left space, about midway between the mammary and parasternal lines (the former being a vertical line drawn through the nipple, the latter being parallel to it, and drawn midway between the nipple and the edge of the sternum), and is formed by the left ventricle. The *right edge* extends from the third right costal cartilage to the junction of the fifth or sixth costal cartilage with the sternum. This edge is convex, the convexity being directed outwards, so that in the third and fourth spaces it is from 1 to 1½ inches from the edge of the sternum: it is formed exclusively by the wall of the right auricle. The *inferior edge* extends in a slightly curved line from the junction of the fifth or sixth right costal cartilage with the sternum to the apex: it is formed by the wall of the right ventricle and the apex of the left. The *left edge* extends from the apex to the level of the third left costal cartilage, where it joins the base: it is formed by the wall of the left ventricle.

Posteriorly the base of the heart is at the level of the fifth dorsal vertebra, and the apex at the level of the eighth.

The groove marking the boundary between the right auricle and ventricle is represented by a line drawn from the junction of the third left costal cartilage with the sternum, downwards and to the right, to the junction of the fifth or sixth right costal cartilage with the sternum.

If the *position of the four cardiac orifices* be marked as in Fig. 11, it will be seen that they are all included within a very limited area of the surface of the thorax. They may be given thus—

Aortic orifice.—Sternum adjoining third *left* cartilage and space.

Mitral orifice.—*Left* half of sternum from fourth to fifth cartilage.

Pulmonary orifice.—Second *left* space and sternum adjoining.

Tricuspid orifice.—*Right* half of sternum from fourth to sixth cartilage.

Two of these, the pulmonary and tricuspid orifices, are comparatively near the chest wall; while the other two, the aortic and mitral, occupy a much deeper position in the chest.

The *left auricular appendix* is opposite the second left space, and to the outer side of the pulmonary artery, which it touches.

The *right auricular appendix* lies behind the right half of the sternum, on a level with the third cartilage.

These and some of the following relations are represented in the frontispieces.

RELATIONS OF THE LARGE VESSELS ABOVE THE BASE OF THE HEART.

The *arch of the aorta* lies behind the sternum, its transverse portion crossing about an inch from the supra-sternal fossa, that is, on a level with the second rib; on the right side it projects somewhat into the second space and under the second cartilage.

The *innominate*, the *left common carotid*, and *left subclavian* arteries arise from the transverse portion of the arch; the first passing upwards to the back of the right sterno-clavicular articulation, and the second to that of the left.

The *superior vena cava* lies to the right side of the arch, opposite the inner ends of the first and second cartilages and intercostal spaces.

The *left innominate vein* is in contact with the upper part of the aortic arch, its superior limit being nearly on a level with the upper edge of the sternum.

The *pulmonary artery* lies behind the sternal end of the second left intercostal space for about an inch.

The *left pneumo-gastric*, *phrenic*, and *superficial cardiac nerves* lie in front of the transverse portion of the aortic arch, and the left *recurrent laryngeal* turns up under and behind it.

Posteriorly the aorta reaches the spine to the left of the body of the fourth dorsal vertebra, and at the lower part of the body of the fifth it becomes the descending thoracic aorta.

Physical Examination.

Inspection of the Precordia.

The precordia is the part of the thorax situated in front of the heart. Normally it is slightly bulged, as compared with the corresponding part of the thorax to the right of the sternum.

Precordial Pulsation ("Apex-beat").—The impulse of the heart against the thoracic wall is normally visible in the fifth intercostal space, about three inches from mid sternum. This is known as the *apex-beat*. In relation to the nipple it is in the space immediately inferior to it, and from half an inch to one inch within it. In the normal condition the impulse of the heart is visible at the apex only. The normal apex-beat is produced by the impulse of the apex of the left ventricle, but the term "apex-beat" is very loosely applied, and is often used to denote the part of the precordia where pulsation happens to be most marked. Thus it results that in many morbid states the so-called apex-beat is not caused by the left ventricle at all, but by the right. In recording cases it would be better if, instead of stating that the apex-beat is in such or such a space, it were stated that pulsation is visible in such and such spaces, while at the same time the position of the pulsation should be indicated in relation to the mammary line, whether within or without it, and also in relation to the sternum.

Abnormal bulging occurs when the heart is much enlarged, or when there is much fluid in the pericardial sac; in the latter the bulging, especially of the intercostal spaces, may be very marked. When the precordia is being inspected, any abnormal bulging above the base of the heart ought to be noted at the same time, but more special reference will be made to this under inspection of the blood-vessels.

Drawing in of the precordia, more especially of the interspaces, during ventricular systole is sometimes present when the pericardium has become adherent to the heart as a result of pericarditis. This requires to be distinguished from the falling in of the intercostal spaces, which occurs during systole in some cases of effusion, and of enlarged and hypertrophied heart.

Irregularity in the time and force of the impulse cannot be investigated so satisfactorily by inspection as by palpation.

Alterations in the Position and Area of the Cardiac Impulse.

Displacement downwards and outwards of the apex-beat is the term used when pulsation is lower and further to the left than normal. The form of expression is, however, too loose, and the position of pulsation ought to be more precisely specified. The condition occurs in hypertrophy and dilatation, especially of the left ventricle. It is present in a still more marked degree in aneurism of the aorta, when the aneurismal sac is large: then there is a true displacement, the heart being pushed downwards and outwards, so that it may even be seen pulsating in the axillary line. It is displaced in the same direction when there is copious pleural effusion on the right side.

Displacement upwards occurs when the heart is pushed up. This is present most commonly in cases of great abdominal distention from tympanitis, ascites, or abdominal tumour.

Pulsation to the right of the sternum occurs from displacement of the heart to the right by a copious pleural effusion on the left side: then its impulse is frequently seen below the right nipple. It is also displaced to the right by partial collapse or contraction of the right lung.

Pulsation of the right ventricle is very frequently present. It is seen in the third, fourth, and fifth left spaces, in the last of these extending outwards to within an inch or so of the apex. This results from thinness of the chest walls, or is caused by dilatation of the right ventricle, which may follow upon mitral stenosis or regurgitation, or may be due to obstruction to the pulmonary circulation, as in emphysema. This last is the least common cause of pulsation in the interspaces mentioned, as in emphysema the heart becomes covered with lung, and in addition is displaced downwards, so that the pulsation is more commonly epigastric. It is of the utmost importance that the position in which pulsation of the right ventricle is to be noted should be thoroughly grasped. It is still taught that pulsation in the epigastrium is the distinctive mark of dilatation of this ventricle. No doubt pulsation in the upper part of the epigastrium does denote dilatation of the right ventricle, and, as has been stated above, it may be the only seat of pulsation in emphysema with dilatation of the right ventricle; but pulsation in the spaces mentioned is equally an evidence of the condition, and is much more frequently present, and indicates a commoner degree than epigastric pulsation. This is the inevitable teaching of anatomical facts, and cannot be too strongly insisted upon, if it is sought to acquire an intelligent comprehension of cardiac changes.

Pulsation in the second left space is not infrequently present. It is sometimes caused by the distended pulmonary artery, at other times by the dilated conus arteriosus of the right ventricle.

Character and Rhythm of Pulsation.

The character of precordial pulsation varies considerably. Besides variations in force, there is an undulatory or wave-like pulsation, which may be present in pericardial effusion or in dilatation of the right ventricle, if the ventricle is in contact with the parietes.

The rhythm of pulsations at different points can be satisfactorily demonstrated by placing small flags on the chest wall. It can then be seen whether the pulsations are synchronous with one another, or what their precise relations are in point of time.

Negative Evidence from Inspection.

By this we mean the absence of positive evidence, as the absence of bulging, or of pulsation. The importance of such negative evidence must not be under-estimated. When no impulse is visible it ought to be stated. Negative evidence is not, however, necessarily of much importance, and this is particularly so in muscular and fat chests, where, in consequence of the thickness of the parietes, there may be no precordial pulsation visible.

PALPATION OF THE PRECORDIA.

The precordia is palpated by placing the hand flat on the chest. By this means we feel the movements of the heart as they are transmitted through the parietes. In the normal condition the systolic impulse against the thoracic wall is felt at the apex, while at the base the shock of the closure of the semilunar valves can be felt when the thoracic wall is thin or the cardiac action excited. When the *organ is feeble* its impulse may only be felt by making the patient bend forwards; when it is *hypertrophied* the measured thud is very characteristic.

Pulsation is felt over a wide area when the heart is enlarged, and it usually corresponds with the area over which it is visible.

Both the systolic impulse and the vibrations set up by the closure of the semilunar valves are felt at the apex in many persons, more especially in those with thin chests, or who are clinically known as of a nervous temperament.

Irregularity in the time and force of the impulse occurs in many morbid conditions, more especially in mitral lesion, and in fatty, debilitated, and dilated hearts. It is also present in some nervous affections.

Thrills are sometimes to be felt over the precordia, either at the apex or at the base. The sensation has been compared to the feeling experienced when the hand is placed on the back of a cat when it is purring; it has accordingly been called *frémissment cataire.* They are most commonly produced at the mitral, the aortic, or the pulmonary orifices. At the mitral orifice they may be systolic, or diastolic, or distinctly pre-systolic, and are felt at the apex: at the aorta or pulmonary artery they are almost invariably systolic, and are felt at the base. They are caused by narrowing of the orifice at which they occur, and for their production the blood has to flow with considerable force. As a consequence of this they disappear if the nutrition of the cardiac muscle becomes much impaired, and reappear with its rehabilitation.

Cardiography.—The movements of the precordia can be registered by means of the cardiograph. Tracings obtained by this graphic method show the sequence and duration of the cardiac movements; they give clear indications of such abnormal vibrations as those causing reduplication of the sounds of the heart, but render less distinct evidence of the vibrations producing murmurs. Cardiography has not yet been able to render any real service to clinical medicine, and no more need be said about it in this place.

PERCUSSION OF THE HEART.

The percussion of the heart is divided into superficial and deep, according as it is desired to delimit the part of the organ uncovered by lung, or to find the precise size of the organ itself. The former is the *superficial* or *absolute dulness*, the latter the *deep* or *relative dulness*.

Superficial Dulness.

The superficial dulness is the part of the heart uncovered by lung, and lies within the notch in the left lung, and between it and the edge of the sternum. What has really to be done in defining the superficial dulness is to find the edge of the left lung. Percussion must be *very light*, as the edge of lung overlying the heart is very thin at its margin, and all trace of clearness in the sound must disappear before it is inferred that the margin of the lung is passed. Normally the superficial dulness extends from the fourth to the sixth left costal cartilage, and extends outwards to about the junction of the cartilages with the ribs. The part of the heart under the sternum which is uncovered by lung is not readily defined, and can only be made out by comparing the percussion sound on the sternum above and below it.

The *clinical value* of the superficial dulness is not great, and many clinicians have entirely abandoned its investigation. When, however, the heart is much enlarged, or when there is much pericardial effusion, the area of this dulness is extended as the organ approaches the chest wall and pushes the lung aside. On the other hand, when the volume of the lung is increased, as in emphysema, the area of dulness is diminished, for the edge of the left lung approaches nearer to the middle line and covers more of the heart. Emphysema of the edge produces the same result.

Deep Dulness.

The deep dulness of the heart is, on the other hand, of very great importance and value to the clinician, for by means of it he can determine its size and its position in the thorax. The percussion in this case must be strong, and it requires to be understood that the change in the percussion sound as we pass from lung only to lung with heart below it is at first very slight, indeed almost imperceptible to the untrained ear, although it becomes more distinct the nearer we approach the sternum, and, consequently, as the depth of lung overlying the heart diminishes. Perhaps the greatest difficulty in the percussion of this organ is the acquirement of confidence in our observations, for the organ as it lies in the thorax is covered by a greater extent of thoracic wall than is quite appreciated by clinicians as a rule. It is also to be remembered that the large vessels above the base of the heart, and under the sternum and adjoining cartilages and spaces, act as a solid body to percussion. As a consequence of this, their dulness is continuous with the cardiac dulness, and we make a rule of defining both. The method we use is as follows:—We begin to percuss in the outer half of the first or second space on the left side, and percuss strongly, always moving inwards, until a slight alteration in the sound is perceived, and at that spot make a mark; the second or third and succeeding spaces are percussed in the same manner, always being mindful to begin far out, so as to get a pure lung sound to start from, and in each space a mark is made where dulness begins. If it is desired, a similar proceeding may be followed with the ribs, but as a matter of fact it is superfluous, for if we have a mark in each space (a dot is sufficient) all that is necessary is to join them by means of a line, and thereby a very accurate outline of the left edge of the heart, as it lies in the pericardium, and of the vessels at its base, is obtained. In this way the true apex of the heart is found, a point of importance when auscultation is followed, and which is to be distinguished from the precordial pulsation in other positions, often erroneously called the apex-beat. A like proceeding is adopted to define the right edge of the vessels at the base and the right edge of the heart. With reference to the latter, however, it is to

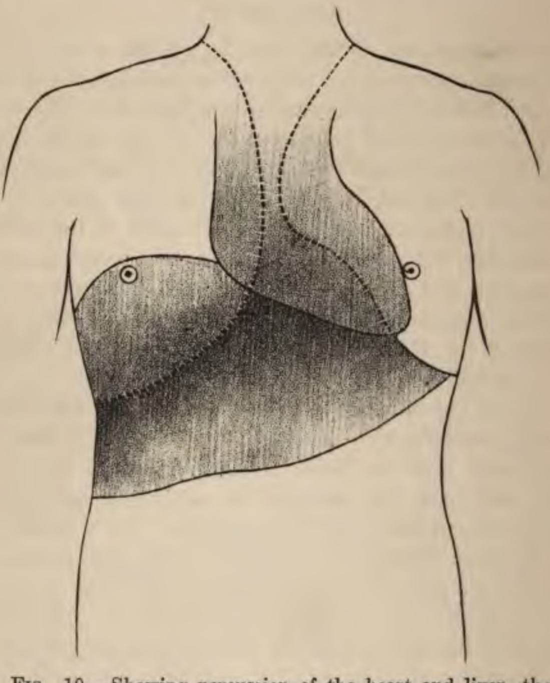


FIG. 10.—Showing percussion of the heart and liver, the degree of shading indicating the degree of dulness. The dotted line indicates the margin of the lungs. (From a photograph.) The liver in this case is larger than usual.

be remembered that the depth of lung between the parietes and the part of heart to the right of the sternum is considerable, and that the difference in sound when we pass from lung only is correspondingly slight. Lastly, there is to be considered the delimitation of the lower border of the organ. If the right and left borders have been completed, a line drawn to join their inferior limits gives this border with sufficient accuracy for all practical purposes. The delimitation of the border by percussion is usually regarded as impracticable, owing to the organ being in such close apposition to the liver. This is, however, by no means invariably the case, for there can often be made out a distinct difference in the character of the dulness over the two organs, which, by observations verified on the dead body, is found to accurately indicate their respective bounds.

The accompanying diagram (taken from a photograph) represents the outline obtained by percussion after the method given above.

The cardiac dulness is increased in area in all conditions in which the organ is enlarged, and when there is pericardial effusion. The dulness given by the vessels at the base is increased, both in intensity and extent, in dilatation and aneurism of the aorta.

The position of cardiac dulness is of course altered if there is any alteration in the position of the organ, as when it is displaced to the right side, or downwards and outwards, or downwards only, or upwards. These have also been referred to under inspection and palpation. It is unnecessary to dwell in detail on the position of the dulness in all these, but the displacements must be kept in mind, and accuracy in determining their limits will be attained by practice.

AUSCULTATION OF THE HEART.

When the stethoscope is placed on the precordia the sounds produced by the heart are heard. They are two in number, and are commonly represented by the two monosyllables *lūb-dŭp.*

The first of these corresponds with ventricular systole, and for purposes of physical diagnosis may be regarded as mainly caused by the closure of the auriculo-ventricular valves, that is, the mitral and tricuspid, and only in an unimportant degree by the sonorous vibration emitted by the contracting muscular substance. At the same time it is to be understood that the strength of the muscle determines the vigour of the contraction, and therefore the vigour with which the auriculo-ventricular valves close.

The second is caused by the closure of the semilunar valves (the aortic and pulmonary), and marks the *beginning* of ventricular diastole.

The first or systolic sound is heard best at the apex and over the right ventricle, and is only faintly audible at the base.

The second or diastolic sound is, on the other hand, best heard over the base of the heart; the aortic second sound being heard best in the second right space, while the pulmonary second sound is heard best in the corresponding space on the left side: it is also audible over the entire sternum, and over the body of the heart as far to the left as the apex. The second sound is normally louder in the aortic than in the pulmonary area.

For convenience in describing auscultatory signs, the precordia is divided into *four areas,* called respectively the mitral, tricuspid, aortic, and pulmonary. The *mitral area* is represented in Fig. 11 as occupying an area about two inches in diameter, the centre being the apex. The *tricuspid area* embraces the area from the third to the fifth intercostal spaces on the left side and the adjoining sternum, as shown in the figure. The *aortic area* is usually given as the inner end of the second or third right rib or second space; in the figure it has been placed partly over the sternum. The *pulmonary area* is the inner end of the second left space.

At the base the first or systolic sound is partly caused by the closure of the auriculo-ventricular valves, but in part by the vibrations of the walls of the aorta and the pulmonary artery, from their sudden tension as the blood is driven into them by their respective ventricles.

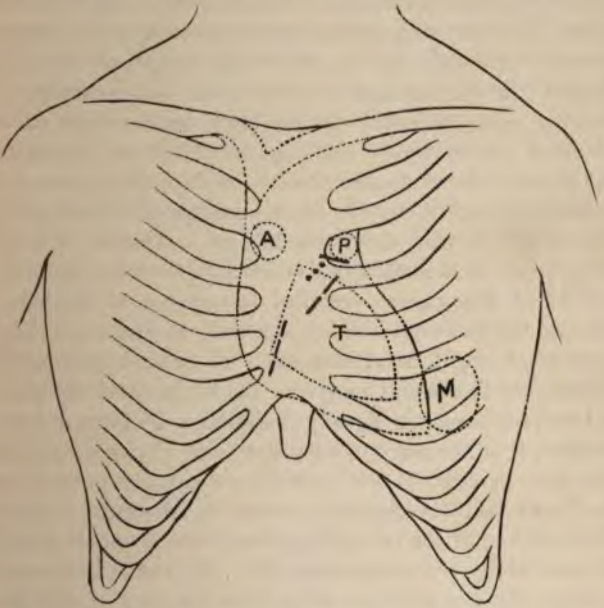


FIG. 11.—Showing precordial areas and the position of the orifices in relation to the surface (latter from Sibson). *A*. Aortic area; *P*. Pulmonary area; *T*. Tricuspid area; *M*. Mitral area. ... Aortic orifice; — Pulmonary; - - - Tricuspid; – – Mitral.

The relative intensity of the two sounds at the apex, over the right ventricle midway between the apex and the edge of the sternum, over the lower half of the sternum, and in the aortic and pulmonary areas, ought to be carefully studied, for, although there is considerable variation in different healthy individuals, valuable indications are sometimes obtained from such observations in abnormal conditions.

Modifications of the First Sound.

A. *Diminution in the intensity of the first sound* as heard at the apex occurs in all conditions in which the strength of the left ventricle is impaired. This is notably the case in all febrile diseases; to a certain extent in cachectic and wasting diseases, although not to the same extent as in febrile diseases; in anæmia and debility, from various causes; and in fatty heart, whether due to fatty degeneration of the muscle of the ventricle or to deposit of fat on the surface and between the muscular fibres. In fat persons, especially women, the sounds may be faint from the thickness of the chest wall overlying the heart, but in persons of this type the strength of the organ is also not infrequently impaired.

It is of the utmost practical importance to be able to estimate the power of the left ventricle by auscultation, and the student ought to avail himself of every opportunity of listening to hearts and analysing the loudness of the sounds at the various points already indicated. To form a correct estimate, it is necessary to auscultate over the *apex*,—not the apex-beat necessarily, for, as has been already pointed out, the "apex-beat" is frequently caused by the right ventricle. The position of the true apex, when there is doubt, must be determined by percussion (page 50). If, instead of auscultating at the true apex, we do so over the part of the chest which covers the right ventricle, the first sound may be loud, although the muscle of the left ventricle is degenerated; this is because the sound produced by the right side of the heart is as a matter of course heard best immediately over it, and because the muscle of the right ventricle may be comparatively strong, although the left is weak. The state of the pulse, as regards both frequency and strength, depends mainly on the frequency and vigour of the contractions of the left ventricle, and it ought always to be examined before a definite opinion is formed as to the condition of the ventricle. The neglect of these precautions leads to many errors. Further, it must not be forgotten that temporary excitement, such as the excitement caused in the majority of persons by being subjected to physical examination, stimulates the heart to increased vigour, and therefore to a louder first sound and a stronger pulse. If, therefore, an opinion be formed by a rapid examination under such circumstances, it will most probably be incorrect. Even when the first sound is so weak as to be inaudible at the apex, the second sound may continue to be distinctly audible there, and may give the erroneous impression of being accentuated. When the first sound is thus inaudible, all that is heard at the apex is of course the dŭp-dŭp-dŭp of the second sound, with a longer or shorter silence in between. Instead of the somewhat grave tone of the second sound, it often becomes clicking in character.

B. *Increase in the intensity of the First Sound.*—This may present two varieties. In one form the sound is long and grave (as distinguished from short and sharp), a like impression being conveyed to the mind of the observer by the character of the impulse communicated through the stethoscope to the head. When the sound has this character, it is followed by an abnormally loud second sound. It occurs when the left ventricle is hypertrophied and its muscle well nourished. It is present most typically in cases of cirrhotic kidneys, and in some cases of aortic stenosis and aortic aneurism.

In the second variety the first sound is short and sharp, and is not of the same diagnostic importance as the first form. It occurs in persons with healthy hearts who have thin chests, and from comparatively unimportant causes, as mental emotion or slight excitement, flatulence or other symptom of indigestion, or a little extra physical exertion. The individuals who are classed as "nervous" frequently present this peculiarity of the cardiac sounds. It also occurs in febriculæ, and may be present at the onset of any acute febrile disease.

C. *Modifications in the tone of the First Sound.*—The first sound is liable to various modifications in tone, some of which may be noted. It may be short and sharp, long and grave, flopping, or impure. The two last are not infrequently mistaken for murmurs, and sometimes indicate a condition in which murmur may be developed later or on exertion.

D. *Alterations in Rhythm.—Reduplication of the First Sound.*—This is best understood by considering it as the first sound with a break or notch in it, as may be represented graphically as follows:—

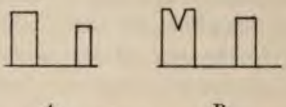


FIG. 12.
Normal first and second sound. Reduplicated first sound.
(After Wyllie.)

There is some difficulty in absolutely determining the cause of the phenomenon, but the two main views entertained as to its mode of production are—(1.) that it is caused by non-synchronous closure of the mitral and tricuspid valves ; (2.) that it is due to non-synchronous tension of the cusps of the same valves. It occurs in lesions of the auriculo-ventricular valves, usually when compensation has failed.

Irregularity presents much variety both in time and strength. No two successive sounds may correspond, or there may be a few intense sounds followed by a number of weak ones. It is a marked feature in many cases of mitral lesion, in fatty heart, and in some cases of debility, especially during or following on acute illness.

Intermittence is the missing of a contraction, and therefore of the sound. It is to be distinguished from irregularity. Its cause is not always apparent, but it may occur in dyspepsia in gouty persons, while at other times it appears to be a neurosis, and in some persons is habitual and of no practical bearing. The number of intermissions should be noted in relation to the number of beats as 1 in 10-12, or whatever the number may be.

Modifications of the Second Sound.

A. *Diminution in the intensity of the second sound* occurs in all conditions where the power of the ventricles is diminished. The blood, under such circumstances, is propelled into the aortic and pulmonary arteries with less vigour, and also in diminished volume, and as a consequence the elastic recoil of these vessels is lessened. It is reasonable to assume that if the ventricles be materially weakened, the volume of blood propelled by each systole must be reduced—that, in fact, the ventricles do not empty themselves to the extent they do normally. That this must be the case is borne out by the results observed in fatty heart where the pulmonary circulation and the systemic venous circulation become engorged. That the intensity of the second sound depends on the elastic recoil of the vessels is abundantly proved by physiological and pathological observations.

B. *Accentuation of the second sound* may have its seat at either of the arterial orifices. The method by which it is determined at which orifice the accentuation occurs is as follows :—The second sound, it will be remembered, is normally louder in the aortic area than in the pulmonary ; when, therefore, it is as loud, or louder, in the pulmonary area than in the aortic, it indicates accentuation of the pulmonary second sound. Accentuation of the aortic second sound can only be estimated by a knowledge of its average loudness under normal conditions.

Accentuation of the pulmonary second sound results from any obstruction to the pulmonary circulation, whether due to valvular lesion at the mitral orifice, or to lesions of the lungs themselves, such as emphysema, &c., which obstruct the capillary circulation.

Accentuation of the aortic second sound depends on obstruction in the arterial circulation, and is a concomitant of chronic renal disease, more especially in its cirrhotic forms ; it also occurs in atheroma, dilatation, and aneurism of the aorta.

C. *Modifications in the tone of the Second Sound.*—In addition to those already referred to the second sound has in some cases a flopping character, which indicates a want of tonicity or of tension in the vessel, usually the result of a weak ventricle with the systemic arteries not either thickened or atheromatous. It is frequently present in febrile conditions.

D. *Alterations in Rhythm.—Reduplication of the Second Sound.*—This may, like reduplication of the first sound, be regarded as the second sound with a break or notch in it, and is graphically represented as follows :—

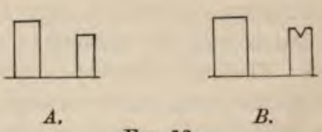


FIG. 13.

Normal first and second sounds. Reduplicated second sound.

(After Wyllie.)

It may, however, be much more marked than this, the second part being distinctly separated from the first, although immediately following it.

It is caused by non-synchronous closure of the aortic and pulmonary valves, and results from alteration in the relative tensions of the aortic and pulmonary arteries. It occurs most commonly in cases where the tension in the pulmonary artery is greatly increased, and where at the same time there has been a proportional lowering of that in the aorta. It is best heard over the sternum at the level of the pulmonary and aortic orifices, but can often be heard over the entire precordia, and very distinctly at the apex.

Irregularity and intermittence accompany similar alterations in the first sound.

Cardiac Murmurs.

Cardiac murmurs or *bruits* are abnormal sounds produced by some morbid condition of the heart. They are divided into exocardial and endocardial.

A. Exocardial Murmurs.

The commonest exocardial morbid sound is *pericardial friction*, the result of pericarditis. This sound is synchronous with cardiac systole or diastole, or with both, and is rubbing, grating, rasping, or creaking in character. It is distinguished from the friction of pleurisy by being synchronous with some part of the cardiac revolution; consequently its rhythm and frequency differ from pleuritic friction, the rapidity of which depends on the respirations, and occurs synchronously with them. Friction is usually first and best heard at the base of the heart. When the intercostal spaces are thin, the friction sound can be made longer and rougher by pressing firmly with the stethoscope; care must be taken, however, not to give the patient pain by this proceeding.

In persons with thin chest walls and a dilated right ventricle, the pulsations of which are well-marked in the interspaces to the left of the sternum, a rubbing sound resembling friction can occasionally be produced by pressing firmly with the stethoscope in an intercostal space. This must not be confused with friction the result of pericardial inflammation.

Pleuro-pericardial friction is the term used to denote the sound produced by the rubbing of the inflamed pleural surface of the pericardium against the inflamed visceral pleura in contact with it. The friction in this condition is, as a rule, only heard during inspiration, and when it accompanies the cardiac systole; it usually disappears with expiration, but it may continue to be *faintly* audible even then with each cardiac systole. Even in the latter case, however, it becomes much louder during inspiration.

If *air and fluid* be present in the pericardial sac, splashing and churning sounds, metallic in quality, are produced, but this is rarely met with, and usually results from puncture from without.

When, as a further result of the pericardial imflammation, *serum is exuded* into the sac, *friction disappears* owing to the pericardium being kept from contact with the heart. When the fluid is reabsorbed or artificially removed the friction reappears.

B. ENDOCARDIAL MURMURS.

Endocardial murmurs are produced at the various orifices of the heart, at the left or right auriculo-ventricular orifice, or at the aortic or pulmonary orifice. They usually result from coarse pathological changes of the valves, or of the endocardium adjoining them, or from a combination of these. Other causes leading to their production will be referred to later.

Time of Murmurs.

The time of murmurs is the first point to be considered. For clinical purposes we may regard a cardiac revolution to consist of ventricular systole and ventricular diastole; and all endocardial murmurs occur during one or other of these. When, therefore, murmurs are spoken of as "systolic" or "diastolic," it is to be understood that it is ventricular systole or diastole that is referred to. At any of the four cardiac orifices two murmurs may occur, namely, a systolic one or a diastolic, or both. Murmurs are *timed* either by placing the hand over the apex-beat, or the finger on the common carotid. The latter is the preferable method. The radial pulse must not be taken, as it is appreciably later in time than the first sound.

Murmurs at the Aortic Orifice.

Taking the murmurs on the left side of the heart first, we begin with those at the aortic orifice.

Aortic systolic murmur is synchronous with the systole of the left ventricle, and therefore coincides in time with the apex-beat and the carotid pulse. It may be caused (1.) by partial adhesion of the cusps to one another; (2.) to thickening and roughening of the cusps, or calcareous deposit at their junction with the endocardium; (3.) not infrequently to a calcareous mass on the endocardium between the base of the anterior cusp of the mitral and the base of the aortic cusps, the region known to anatomists as the intervalvular space of Sibson; (4.) a roughened cusp the result of injury; and (5.) to aneurismal, or even a slight degree of dilatation of the aorta above the aortic valves. In the first three the murmur is the result of obstruction to the current of blood driven from the ventricle into the aorta during its systole; in the fourth it is due to the dilation of the aorta producing a condition analogous to obstruction at the orifice of the artery, and which may be referred to as relative stenosis. In the one case the blood passes through a normal orifice into an abnormally dilated part beyond, while in the other it passes through an abnormally small orifice into a normal part beyond. The latter condition is thus equivalent, from a physical standpoint, to the former. The physical explanation of the production of a murmur under these conditions is, that when a fluid passes through an opening narrower than the space beyond it, a *fluid vein* is produced, and whenever this occurs a murmur results. For further information regarding the formation of fluid veins a work on physics may be consulted.

Aortic diastolic murmur occurs during diastole of the ventricle. It is caused by the blood pouring from the aorta into the ventricle, as a result of the valves not closing the orifice as they normally do. The lesion of the valves which most commonly produces this murmur is shrinking and thickening, but perforations or rupture, and vegetations or calcareous nodules on them, also produce it. Large vegetations or calcareous nodules prevent the edges of the cusps coming into contact and overlapping, and thus an aperture is left through which the blood escapes into the ventricle.

These are represented in the Figures 14 and 15.

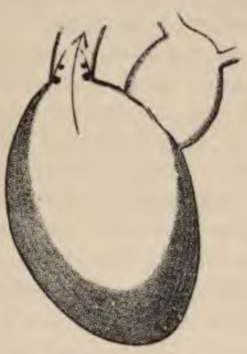


FIG. 14. — Showing aortic systolic murmur from stenosis. Mitral valve closed.

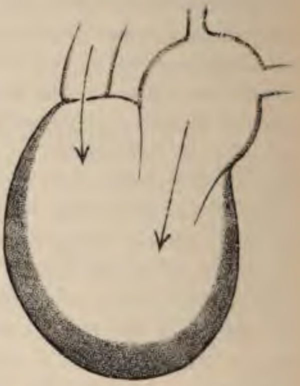


FIG. 15. — Showing aortic diastolic murmur from incompetence. Ventricle filling from auricle at same time.

The arrows indicate the direction of the blood-stream.

Murmurs at the Mitral Orifice.

These occur either during ventricular systole or ventricular diastole.

Mitral systolic murmur is produced by part of the blood in the ventricle being driven during systole, into the auricle, owing to the auriculo-ventricular orifice being imperfectly closed by the mitral valve. This commonly results from shrinking and thickening of the cusps, the cusps being either too small to close the orifice, or too rigid to admit of their sufficiently fine adjustment. It also occurs where there is no disease of the valve, but when the ventricle is dilated and enfeebled; then either the orifice is dilated so that the cusps are too small to close it, or the muscle, owing to weak action, does not sufficiently contract the orifice to enable the valve to close it.

Mitral diastolic murmurs occur during the diastole of the ventricle, that is, when the blood is flowing from the auricle into the ventricle. They are caused by narrowing of the orifice through which the blood passes. The most common lesion producing the condition is adhesion of the anterior and posterior mitral cusps to each other along their lateral edges. By this adhesion the channel for the blood is necessarily much contracted. The valve is in addition thickened and more rigid than normal, and the aperture in it may be exceedingly small. These murmurs are subdivided into—

a. Diastolic (or post-diastolic).

b. Pre-systolic (or auriculo-systolic of Professor Gairdner).

a. The *diastolic mitral* is unfortunately also called post-diastolic. This term is wrong, and here the murmur will be referred to by the former term. The murmur as a rule immediately follows the second sound; it may, however, accompany the second sound as well as follow it, but it does not occupy the whole time of ventricular diastole. The fact that the murmur follows the second sound led to its being called post-diastolic, but from what has been said it will be understood that it occurs not after but during diastole, although it occurs after the second sound; and the second sound, it will be remembered, occupies only a short space of time at the beginning of diastole. The murmur occurs during the time that the blood ought to flow noiselessly from the auricle into the ventricle, and before the auricle contracts. It is comparatively rarely present even when there is marked narrowing of the blood-channel: the reason of this is not quite evident, but it probably depends, in part at least, on the degree of vigour in the suction power of the ventricle.

b. The pre-systolic mitral murmur occurs, as its name implies, before the systole of the ventricle, and at that part of the cardiac cycle when the auricle contracts. It has therefore been called by Professor Gairdner *auriculo-systolic;* here, however, the term pre-systolic will be used. Its production at the particular moment is attributed to the greater energy with which the blood is flowing into the ventricle owing to the contraction of the auricle.

Systolic and diastolic mitral murmurs are represented in the following diagrams.

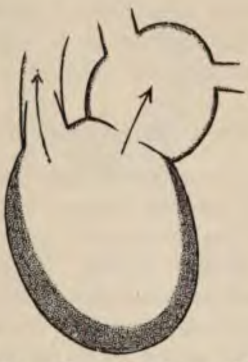


FIG. 16. — Showing mitral systolic or regurgitant murmur from failure of valve to close orifice. The blood current ought to be into the aorta only.

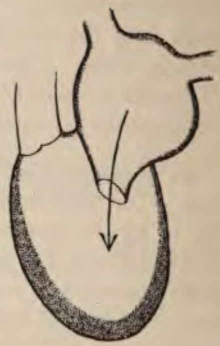


FIG. 17.—Showing diastolic or pre-systolic mitral murmur due to adhesion of mitral cusps.

The arrows indicate the direction of the blood-stream.

Having considered the murmurs which may be present on the left side of the heart, at the aortic and auriculo-ventricular orifices, it is only necessary to refer briefly to those at the corresponding orifices on the right side. These are—

Murmurs at the Orifice of the Pulmonary Artery.

Organic murmurs at this orifice are exceedingly rare; they are systolic and diastolic in time, and are due to lesions similar to those described as causing murmurs at the orifice of the aorta. When they do occur they are not infrequently congenital, and are associated with other lesions, such as patent ductus arteriosus, open foramen ovale, or imperfect closure of the ventricular septum. What are known as *functional murmurs* in this artery are always systolic in time, but they will be referred to more fully hereafter.

Murmurs at the Right Auriculo-ventricular Orifice.

The Tricuspid.—Theoretically there may be three murmurs here, as at the corresponding orifice on the left side, but as a matter of fact diastolic and pre-systolic tricuspid murmurs are of extreme rarity, while systolic murmur is very common. When the two former are present, they are due to lesions similar to those producing corresponding murmurs at the mitral orifice,—that is, to stenosis. Systolic murmur is due to incomplete closure of the orifice, either from shrinking and thickening of the valves or from dilatation of the orifice : the latter is the more common.

The position of murmurs in the cardiac cycle may be studied in the diagrams on the following page. Fig. 18 represents the normal cycle, half the circle representing ventricular systole, the other half ventricular diastole, the closure of the semilunar valves and the contraction of the auricles being included in the latter.

Point of Maximum Intensity and Direction of Propagation of Endocardial Murmurs.

The precordia is divided, as has already been indicated, for purposes of auscultation, into four areas. These areas are respectively aortic, pulmonary, tricuspid, and mitral. They indicate the sites at which murmurs produced at the respective orifices are heard best. Although they are given here, it is better to follow the directions given in the following pages, and to trace more in detail the areas over which murmurs are heard.

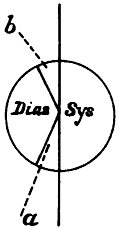


FIG. 18.—Diagram showing ventricular systole and diastole; included in the diastolic half of the circle is—*a.*, the closure of the semilunar valves, *i.e.*, the second sound; and *b.*, the systole of the auricle.

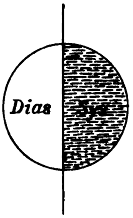


FIG. 19.—Position in cardiac cycle of systolic murmur at any orifice.

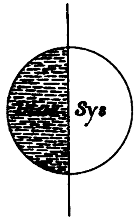


FIG. 20.—Position in cardiac cycle of diastolic murmur at aortic or pulmonary artery.

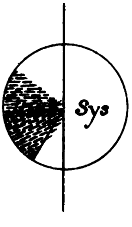


FIG. 21.—Position in cardiac cycle of diastolic murmur at mitral or tricuspid orifice.

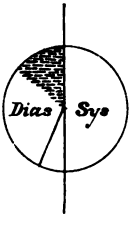


FIG. 22.—Position in cardiac cycle of pre-systolic murmur at mitral or tricuspid orifice.

The shaded part represents the murmurs.

The point where a murmur is heard loudest is known as *the point of maximum intensity*, and the several murmurs have each their distinctive point or seat of maximum intensity. The various murmurs also tend to be propagated in definite directions: this is known as the *direction of propagation*. These two can best be considered together. The general law as regards the propagation of murmurs is that they are propagated best in the direction of the current which produces them. They are also propagated by the sternum, by the walls of the heart, and by solid bodies or pulmonary cavities in proximity to the heart and aorta.

Aortic systolic or obstructive murmur is usually described as having its point of maximum intensity on the second right rib or space,—the "aortic area," as it has been long called. This has led to much confusion and to many errors, and here we propose to depart slightly from the classical position. The murmur when present is always heard over the sternum at the level of the second cartilage, and this is the place where it is best to listen for it. The extent of area over which it is propagated depends mainly on its loudness. When very loud, as it often is, it is distinctly audible over every part of the thorax, both anteriorly and posteriorly; this has to be clearly borne in mind, for unless it is, the fact that it is audible at the apex, for instance, is often mistaken by the student as evidence of the existence of a mitral systolic murmur in addition to the aortic systolic. The murmur, however, is not invariably as loud as has just been indicated, and when this is the case it is conducted or propagated in more definite directions. As has already been said, it is heard over the sternum about the level of the second cartilage. It is propagated—(1.) *Upwards* by the blood-stream, and is, as a rule, heard as distinctly over the entire manubrium as it is at the level of the second rib. By the same means it is conducted into the carotids and heard in them; but, if the murmur be faint over the sternum, it may be inaudible over them. (2.) *Downwards* (*a.*) by the sternum, the bone acting as a good conductor of sound, but, unless the murmur be a very loud one, it diminishes markedly in intensity from about the level of the second space; and (*b*.) by the heart itself, which acts as a conductor of the sound, so that it may be audible over the precordia to the left of the sternum, as far out as the apex.

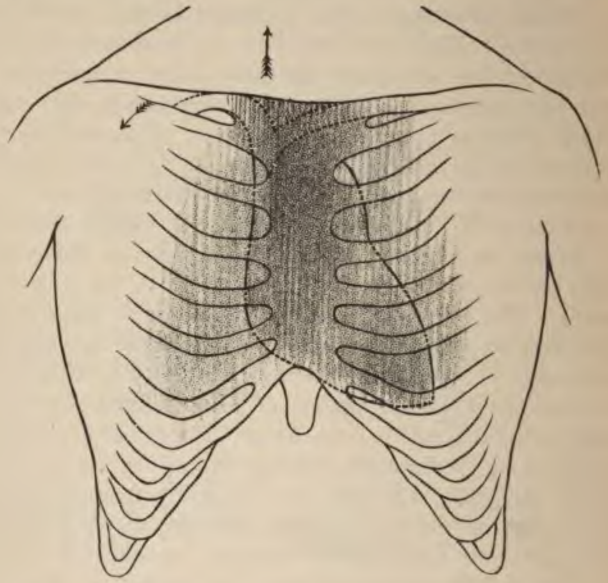


FIG. 23. — Showing area of audition of aortic systolic murmur. The degree of loudness is indicated by the depth of the shading, and the arrows the direction of propagation of the murmur into the arteries.

The above diagram illustrates this.

Aortic Diastolic Murmur or the Murmur of Aortic Incompetence.—The point of maximum intensity of this murmur is over the sternum, the precise point varying in individual cases. The most common points, however, are (*a*.) the sternum at the level of the second cartilage or second space; and (*b.*) the lower part of the sternum. The murmur is as a rule, however, heard all over the sternum, but especially from the level of the second cartilage downwards, while it is usually not nearly as loud over the manubrium: this it will be noted is the reverse of what happens in aortic systolic murmur. When it is heard over the lower part of the sternum, it frequently attains its maximum loudness at or near the base of the xiphoid. In addition to being propagated down the sternum, the murmur may be propagated by the walls of the heart, and heard over the precordia to the left of the sternum. It may, however, be louder over the apex of the left ventricle than at any point between the apex and the edge of the sternum. This is explained by the regurgitant stream carrying the sound with it, and, impinging on the wall of the ventricle, communicating its sound vibrations to it, and thence to the chest wall. Occasionally a diastolic aortic murmur is only audible at the apex of the left ventricle: this is rare, but it undoubtedly occurs. It has been suggested that the phenomenon is due to the left postero-lateral cusp only being affected, and that thereby the direction of the regurgitant stream to a more backward course is determined.

Mitral Systolic Murmur or Mitral Regurgitant Murmur.—This murmur has its point of maximum intensity at the apex of the heart. The area of its audition may be the size of a crown-piece, or even smaller; when, however, it is moderately loud, it tends to be propagated outwards and upwards towards the axilla, and may even be traced as far as the angle of the left scapula. The extent to which it is propagated in this direction depends not only on its loudness, but also on the degree of enlargement of the ventricle, and probably on the transverse capacity of the thorax; the larger the heart and the smaller the transverse capacity of the thorax the more nearly does the heart approach the chest wall in the infra-axillary region, and consequently the greater the area over which the murmur is audible.

It is commonly taught that this murmur is also propagated inwards, and is heard even over the sternum. This is an error which has done much to make the auscultation of the heart one of the most difficult sections of physical diagnosis for the student. In many cases it may be noted that a systolic murmur audible at the apex, and even moderately loud there, is not heard a little way to the right of the apex, and is absolutely inaudible over the sternum and for some distance to the left of it: in these cases there is no reason why the murmur should not be heard in these positions, were it true that mitral systolic murmurs were, as a rule, propagated towards the sternum. In another class of

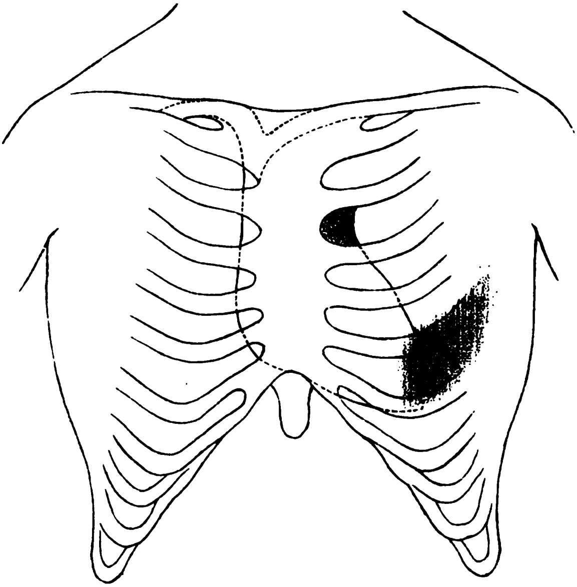


FIG. 24.—The shaded parts indicate the area of audition of mitral systolic and of pulmonary systolic murmurs.

case a systolic murmur is audible over the apex, over the precordia from the apex inwards, and also over the *lower* part of the sternum. Now, when this is the case, there should be no doubt that in addition to a mitral systolic there is also a tricuspid systolic murmur. That there is tricuspid reflux is often proved by the distention and pulsation of the veins at the root of the neck, so that the evidence of the contention that there is a tricuspid as well as a mitral murmur appears to us to be based on accurate observation and correct deduction. From this it will of course be understood that a mitral murmur is never heard over the base of the sternum.

Mitral Diastolic and Mitral Pre-systolic Murmur.—Both these murmurs have their point of maximum intensity exactly over the apex, and are quite localised; so marked indeed is this the case, that at a distance of one inch to either side of the apex they are, as a rule, quite inaudible. A murmur, believed to be a mitral diastolic, is sometimes audible between the third and fourth left cartilages only.

Pulmonary Systolic Murmur.—This murmur has its point of maximum intensity over the pulmonary artery in the second left intercostal space close to the edge of the sternum. It is, as a rule, a localised murmur, and is rarely if ever heard beyond one inch from the sternal edge, or over the sternum adjoining the space, or over the cartilage above or below it. It is frequently present, more especially in anæmia and debility, and it does not, as a rule, denote organic disease at the orifice of the vessel. It will be referred to more fully when functional or anæmic murmurs are considered. Pulmonary systolic murmur is very rarely due to organic lesion of the valves, and then it is commonly congenital and associated with other lesions, as has been already mentioned at page 65. When due to organic disease it is much louder, and, while attaining its point of maximum intensity over the orifice of the vessel, it may be widely propagated and audible over the whole precordia and the whole length of the sternum: it is not, however, propagated into the carotids, and this serves to distinguish it from loud aortic systolic murmur.

The preceding diagram, Fig. 24, shows the position of the pulmonary systolic murmur.

Pulmonary Diastolic Murmur.—This murmur is analogous to aortic diastolic murmur. Its point of maximum intensity is either over the orifice of the vessel or at the lower part of the sternum. Its direction of propagation is down the sternum and over the right ventricle to the left of the sternum. It is very rare, and when present may readily be mistaken for an aortic diastolic murmur. If it were louder over the right ventricle to the left of the sternum than at the apex of the left, it would aid in its differentiation.

Tricuspid Systolic Murmur.—At one time this was regarded as a very rare endocardial murmur. It is no longer so regarded, and there can be no doubt that it is really one of the commonest. Its point of maximum intensity is generally stated to be at the base of the xiphoid, but this is not invariably the case.

The murmur may be only heard in the spaces from the third to the sixth left costal cartilage, and it may be of equal intensity in all of them. When most typical, however, it is heard over all that part of the thorax which covers the right heart, that is to say, it is heard from the septum of the ventricles inwards to the sternum over a triangular area, as represented in Fig. 25, the left boundary of which is formed by a line drawn from the third left cartilage to about one inch within the apex, the inferior boundary being formed by the lower border of the right ventricle. It is also audible over the sternum adjoining this area, and to the right of the sternum in the third, fourth, and fifth spaces, for from one inch to an inch and a half from the right edge of the sternum. When the murmur is thus heard over the whole right heart it commonly attains its maximum intensity over the lower third or two-thirds of the sternum. The position of the tricuspid systolic murmur is thus what might be expected from the relations of the right heart to the thoracic wall. It is heard to the right of the sternum, because the right auricle extends to the right of the sternum; the regurgitating stream carries the sound with it into this chamber, and is propagated through its wall to the thoracic wall. Some other points bearing on this subject will be referred to under anæmic murmurs.

Tricuspid Diastolic and Pre-systolic Murmur.—Both these murmurs are extremely rare. Their point of maximum intensity ought to be on the sternum over the tricuspid orifice, and they ought to be propagated towards the apex of the right ventricle.

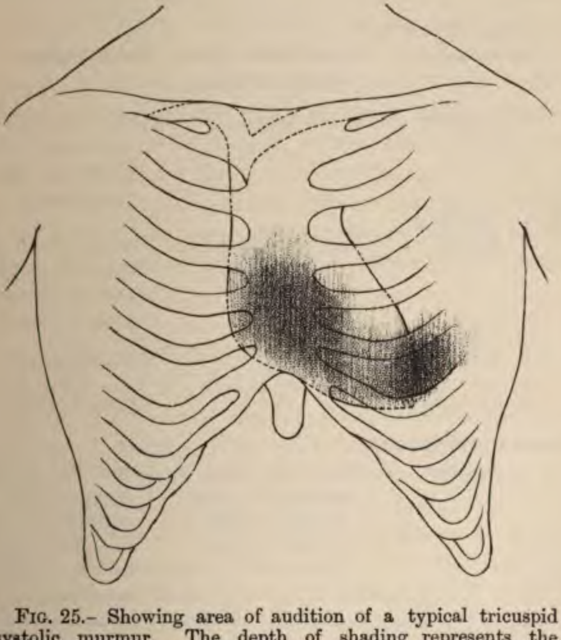


FIG. 25.- Showing area of audition of a typical tricuspid systolic murmur. The depth of shading represents the intensity of the murmur.

SUMMARY OF PRECEDING.

	Point of Maximum Intensity.	*Area of Audition and Direction of Propagation*
1. AORTIC MURMURS.		
A. Systolic, . .	Base of sternum.	Over whole sternum; into carotid artery; over whole precordia; and sometimes over whole thorax, anteriorly and posteriorly.
B. Diastolic, .	Sternum at level of second cartilage, or lower part of sternum.	Over whole sternum, but especially over lower three-fourths; at apex; over whole precordia to left of sternum.
2. PULMONARY MURMURS.		
A. Systolic, . .	Second left space, close to sternum.	Not propagated.
B. Diastolic, .	Second left space.	Down sternum and over right ventricle to left of sternum. (Very rare.)
3. MITRAL MURMURS.		
A. Systolic, . .	Apex of left ventricle.	Over area round nipple; outwards towards axilla; round to angle of scapula.
B. Diastolic, .	Do.	Not propagated.
C. Pre-systolic,	Do.	Strictly localised.
4. TRICUSPID MURMURS.		
A. Systolic, . .	Over ower third of sternum, or in adjoining spaces to left of sternum.	Lower two-thirds of sternum; over right ventricle to left of sternum; over right auricle to right of sternum; over manubrium.
B. Diastolic and Pre-systolic.	Lower half of sternum.	Right ventricle to left of sternum.

The Tone, Quality, or Character of Murmurs.

The general law regarding the tone of murmurs is that direct or obstructive murmurs—that is, murmurs produced by obstruction at any of the orifices—are rough in character, while indirect or regurgitant murmurs are soft and blowing in character. This cannot, however, be regarded as of much diagnostic significance, although in many cases the difference is well marked. Some murmurs are distinctly musical in character, depending probably on some incidental element in the lesion, the nature of which it is usually impossible to do more than guess.

There is, however, one murmur which it is necessary to refer to more specially, and that is the *mitral pre-systolic ;* it is a rough murmur, and runs up to the first part of the systole, that is, to the first sound: it may be compared to the sound produced by pronouncing *wrup.* It is frequently confused with a systolic murmur which occurs very commonly in mitral stenosis, and which ends with an accent, the accentuated part being probably produced by the somewhat delayed flap of the rigid valve. This special modification of the systolic murmur is quite diagnostic of mitral stenosis, but it has nevertheless to be distinguished from the pre-systolic murmur.

The Intensity or Loudness of Murmurs.

This varies within wide limits. The loudest murmurs are usually aortic in origin. It is a point, however, the clinical significance of which is apt to be misunderstood. It necessarily depends upon two factors—(1.) the nature or extent of the lesion, and (2.) the vigour with which the blood is driven through the affected orifice. In systolic murmurs, especially at the aortic orifice, great loudness is not of the same serious import as a proportionally loud diastolic murmur at the same orifice. In some cases this is equally true of mitral murmurs. Loud systolic murmurs, however, always imply that the ventricle is acting forcibly, and is therefore well nourished. The converse is equally true, and thus it is that when the cardiac muscle is enfeebled or degenerated, murmurs become less distinct, and may even disappear, reappearing when the ventricle regains power under treatment. The *reappearance or intensification of murmurs* is thus in many cases of good omen. The murmur which of all others is most prone to disappear and reappear is the pre-systolic mitral. This depends not only on the strength of the left auricle, but also, we think, on the varying degree of distention of the right ventricle, for when the right ventricle is more distended than usual the left apex is pushed backwards by it, and thus away from the chest wall, and the murmur is not then conducted to the thoracic wall.

Multiple Murmurs and their Differentiation.

Several murmurs are frequently present in the same patient, and it will be of assistance to indicate some of the combinations which commonly occur, and the method by which they may be differentiated from one another.

In auscultating the heart the system which is generally in use is to begin by listening at the apex. We, however, have found it better to begin by listening at the base, over the manubrium, and at the level of the second cartilage.

A systolic murmur in the former position, and a diastolic in the latter, indicate that the murmurs are probably aortic in origin. When no murmur is present it may be concluded that the aortic orifice and its valves are normal. The advantage of beginning at the base is thus apparent, for it can be at once decided whether or not one of the most commonly affected orifices is the seat of lesion. From the base we next listen over the lower third of the sternum. If murmurs have been heard at the base, they will probably, but not necessarily, be heard here also; but we shall suppose that there was no murmur heard at the base, but that a systolic one becomes audible over the lower part of the sternum;

when this is the case the murmur is a tricuspid systolic one. It may be followed to the left of the sternum, over the right ventricle, but is lost before we reach the apex. If, however, as most frequently happens, there is a mitral systolic murmur in addition to the tricuspid, we find that as we near the apex the systolic murmur becomes louder, and that it may even be followed outwards beyond the apex towards the axilla. The annexed figure represents a case of this kind. If there is no tricuspid systolic murmur, but only a mitral systolic, there is no murmur over the sternum or to the left of it, until

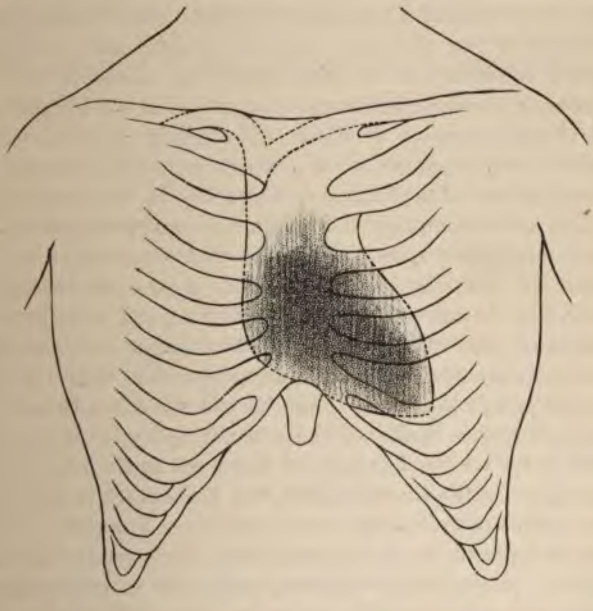


FIG. 26.—Representing a systolic tricuspid and systolic mitral; the deeper degree of shading indicates the points of intensity of the murmurs.

we near the apex, and the murmur is louder an inch or so to the left of the apex than it is at the same distance to the right of it.

We shall take another common condition, in which the differential diagnosis is often very difficult, namely, a case in which there is a systolic murmur heard at the manubrium, all down the sternum, and to the left of the sternum as far as the apex, and even beyond it. Here there is no doubt of one point, namely, that there is a systolic aortic murmur, but the murmur is also audible in the tricuspid region and in the mitral area, and the question which has to be decided is whether or not there is a separate tricuspid and a separate mitral murmur. It is quite impossible to decide whether there is a tricuspid murmur; we may be able by the presence of venous pulsation to affirm that there is tricuspid reflux, but the two murmurs when present cannot be separated by auscultation. The further question is—Is there a separate mitral systolic murmur? There are several rules which help us to decide this question. If the systolic murmur audible over the sternum becomes fainter as the stethoscope is moved to the left over the right ventricle, and again becomes louder or distinctly alters in tone at the apex and beyond it, we may conclude that there is an independent mitral systolic in addition to the aortic. Another aid which we have found to be of service is, that if the murmur is louder at the apex than it is over the left edge of the heart in the space above the apex, or the rib above that, the probability is that there is a mitral as well as the aortic murmur. The reason of this last is apparent, for if the murmur at the apex be the aortic systolic propagated downwards by the left ventricle, there is no reason why it ought not to be as audible at any point along the left edge of the heart as at the apex. In some cases, however, and especially in those in which the aortic murmur is very loud, it is quite impossible by auscultation to determine whether there is a separate mitral. It may, however, be with accuracy assumed that there is mitral reflux if there be evidence of pulmonary congestion and heightened tension in the pulmonary artery.

Diastolic murmur of aortic origin audible at the apex is readily distinguished from mitral diastolic, as the former is a longer murmur than the latter, and occupies the whole time of ventricular diastole, including the time occupied by the second sound, which it may entirely mask and render inaudible.

The three murmurs which have their origin at the mitral orifice may all be present in the same case, but this is not so common as the union of pre-systolic and systolic.

Further Points in the Diagnosis of some Cardiac Conditions.

Mitral Stenosis.—In many cases of mitral stenosis, neither a diastolic nor a pre-systolic murmur is present, and yet the diagnosis may be made with practically absolute certainty. The presence of a systolic murmur ending with an accent has been already referred to as indicative of this lesion. In some cases, however, we have not even this present as a guide, and then the phenomena on which the diagnosis depends, are—(1.) great irregularity in the force and rhythm of the first sound and of the cardiac impulse; (2.) a similar irregularity in the force and rhythm of the pulse, and a great disproportion between the number of cardiac impulses, as felt over the precordia, and the radial pulsations, the former being often nearly twice as numerous as the latter; (3.) marked reduplication of the second sound; (4.) the presence of a systolic murmur at the apex, which may, however, only be heard occasionally, or may not be heard at all when the symptoms are at their worst; and (5.) the distention of the right side of the heart as evidenced by the diffuse heaving over it, the presence of a soft tricuspid murmur, and the distention of the veins at the root of the neck.

It is as well to warn the student that it is often very difficult and sometimes quite hopeless to satisfactorily analyse the sounds in mitral stenosis. This results from the multiplicity of sound producing factors in such cases. Among these may be mentioned a possible, although a questioned, non-synchronous action of the two ventricles; at least it is held by some observers that there are contractions of the right ventricle in which the left ventricle does not seem to participate. There is an occasional loud sound, and a strong systole which can be felt in the carotid pulse, followed by a series of short and irregular sounds, none of which have a corresponding pulsation in the carotid, and which may be caused, some by reduplication of the second sound, others by abortive ventricular contractions, and still others possibly by auricular contractions; but to which cause each sound is to be relegated is, we fear, beyond the discriminative faculty of the human ear. Notwithstanding these difficulties, which are mainly matters of scientific interest, the diagnosis can, as we have said, be made from the very difficulties of the analysis of the auscultatory phenomena. As the heart regains its equilibrium, and distressing symptoms disappear, the analysis of the sounds becomes, however, practicable.

Fatty and Dilated Heart.—In this condition percussion shows the heart enlarged, but not necessarily to any very great extent, the dulness often not reaching beyond the nipple line. The auscultatory evidence is mainly or purely negative. The first sound at the apex is either inaudible or extremely feeble, while the second sound continues more or less distinct: beyond the apex the first sound is quite inaudible. It may, however, be audible over the right ventricle, especially over the sternum, but the sound here is certainly caused by the right and not the left ventricle. In many cases the cardiac action is irregular, and there may be frequent intermissions. Further evidence of the weakness of the ventricle is found in congestion of the lungs from the engorgement of the pulmonary circulation; and perhaps of the systemic venous system also, œdema of the lower limbs being frequently present.

There is sometimes a question of diagnosis, between mitral stenosis and fatty dilated heart with mitral regurgitation, owing to the irregularity which may be present in the latter as well as in the former. The irregularity, in cases of equal gravity as estimated by the symptoms, is, however, very much more marked in mitral stenosis.

Secondary Results of Valvular Lesions.

To understand the results of valvular lesions, it is necessary to have a very clear mental picture of the circulation, and to be able to call it up without effort.

The general law which may be laid down is, that hypertrophy and dilatation occur in the chamber immediately behind the affected valve, and that, as the case advances, the effect tells further back, step by step. Let us illustrate this. Take first aortic stenosis: the result of this lesion is that the left ventricle, which is the chamber immediately behind the affected valve, hypertrophies. So long as the heart muscle is well nourished the hypertrophy is able to overcome the increased resistance, and there is a state of equilibrium or of *compensation*, but, if the nutrition of the muscle be not sufficient for the extra strain, the muscle relaxes and the ventricle becomes dilated. The dilatation tends to increase because the obstruction continues and the condition of impaired nutrition is prone to recur. The impaired nutrition may be the result of extra strain thrown on the heart, which is at the time nourished to its utmost limits; for it has always to be borne in mind that there are limits to increase of nutrition, however wide these limits may be. Whenever this impairment of nutrition or this extra strain (and they may be held as analogous as regards results) supervenes, dilatation results, and thus it is that rest becomes such an important measure of treatment when failure occurs. As a result of repeated loss of equilibrium between the obstruction and the force behind it, the ventricle becomes more and more dilated, and as a consequence the mitral orifice is enlarged and the valves are no longer capable of closing it: this leads to mitral regurgitation. Mitral regurgitation leads to dilatation and hypertrophy of the left auricle. This after a time leads to engorgement of the pulmonary circulation, and to increased tension in the pulmonary artery, indicated by accentuation of its second sound. Engorgement of the pulmonary circulation leads also to the physical signs of bronchitis from the bronchial vessels sharing in the engorgement, owing to their connection with the pulmonary veins. The engorgement also leads to the effusion of fluid into the pulmonary alveoli, producing crepitations; or into the pleural cavity, giving rise to hydrothorax. An early result of the increased tension in the pulmonary artery is that the right ventricle hypertrophies, and after a time dilates. The dilatation increases, as the dilatation of the left ventricle increased, from the obstruction to the outflow of its blood, and following this there is enlargement of the tricuspid orifice with failure of the valves to close it, with, as a result, dilatation and hypertrophy of the right auricle. The final stage is engorgement of the whole venous system, and, as a consequence, effusion of fluid from the capillaries into the tissues and serous cavities, and the establishment of general dropsy or anasarca.

If, in addition to stenosis, there is also regurgitation at the aortic orifice, or if regurgitation occur alone, there is a further dilating force, for there is during diastole not only the normal quantity of blood flowing into the ventricle from the auricle, but in addition a quantity flowing backwards into it from the aorta. The ventricle has to accommodate itself to this increased quantity of blood, and it can only do so by dilating. Here, too, however, a certain degree of dilatation and of hypertrophy maintain the circulation in equilibrium; but extra strain or impaired nutrition are apt to occur, and dilatation increases with its consequent evils, and there is what is known as *failure of compensation.*

From the foregoing it will be easy to trace the changes which follow in the course of time on any given valvular lesion, and they need not be individually detailed.

The general result of all valvular lesions is that the quantity of blood in the parts in front of the lesion tends to diminish, while the quantity behind it tends to accumulate,—in other words, the tension in the arterial system falls, while that in the venous system rises.

It cannot be too strongly impressed upon the student that, while his first task is to master the auscultatory evidence of various valvular lesions, his further and even more important duty is to be able to *distinguish between compensation and failure of compensation*, and to estimate the degree of failure and the power of regaining what may have been lost. He must on no account assume that the presence of cardiac murmurs necessarily demands therapeutic measures, or implies that his patient's death-warrant is to be promptly sealed. On the other hand, many cases in which there are no murmurs require the utmost care and skill to be directed to the treatment of the heart.

The Murmurs present in the Circulation in Anæmia and Debility.

The murmurs present in anæmia are known as *functional or hæmic murmurs*. The first term is used to imply that they are not due to organic structural changes of the valves, and the second that they are due to the condition of the blood. They are usually classed as being produced in the veins, the arteries, and the heart.

1. *Venous Murmurs.*—If the stethoscope be placed over the right jugular vein between the origins of the sterno-cleido-mastoid muscle, a loud continuous murmur of a humming or blowing character is heard in most cases of anæmia and chlorosis. It received the name of *bruit de diable* from its resemblance to the sound made by a French toy of that name. In some cases it is distinctly squeaking in character, more especially if the cardiac action be rapid from the excitement due to examination or to any little special exertion immediately preceding examination. Although the murmur is

continuous it commonly presents distinct periods of greater loudness, which vary in time in different cases; in some the periods of greater intensity being synchronous with the cardiac movements, in others with the respiration. It is heard best if the patient's head be turned to the left, and the chin be at the same time raised. The physical conditions leading to its production have not been satisfactorily defined. The two factors upon which most stress has been laid in the attempt to explain it are, first, the abnormal physical characters of the chlorotic blood, and, secondly, the production of a fluid vein. For the production of the second of these it is necessary that the blood should pass from a narrow into a wider part of the vein. In anæmia it is assumed that, owing to diminution of the total volume of the blood, the veins contract, but that the lower part of the jugular vein cannot so contract owing to its attachments to the adjoining fascia, and that in this way the relatively wider part is obtained and the murmur is produced. The explanation is unsatisfactory because there is no evidence that in chlorosis there is a diminution in the total volume of the blood, and the statement based upon it, namely, that the veins contract to adapt themselves to their lessened contents, can hardly be accepted without further proof than is at present forthcoming. On the other hand, from the facility with which this murmur can be produced in chlorotic patients by placing the head in the position indicated above, it would seem that interference with the lumen of the vein from traction upon it, coupled with the alteration in the physical characters of the blood, may be the more important factors in its production. The altered character of the blood would appear to be a factor, for the murmur is not produced in ordinary cases by the position of the head which so readily produces it in chlorosis. This murmur is sometimes present over the upper part of the sternum, and its presence here is difficult to explain. A murmur of a similar character may be heard over the torcular Herophili and over the orbit, the stethoscope being placed over the occipital protuberance or the closed lid. The systolic accentuation when present can only be accounted for on the assumption of a backward current during ventricular or auricular systole, and there is reason to accept this, from the fact of the venous fulness and pulsation which is present in these cases when the patient is in the recumbent position.

2. *Arterial Murmurs.*—An aortic systolic murmur, having its origin at the orifice of that vessel, is usually described as being present, but we consider, as stated under the following section, that this is, as a rule at least, a mistake. If it is ever present, its mode of production is not satisfactorily explained.

3. *Cardiac Murmurs.*—These are the most important, as they are often mistaken for murmurs due to grave anatomical changes in the valves.

They are always systolic in time, but their seat of origin, and the area over which they are audible, varies in individual instances; they will therefore be considered seriatim.

A. *Pulmonary Murmur.*—This murmur is audible in the second left intercostal space, for an inch to an inch and a half from the sternal edge. It is strictly localised to this area, not being heard over the ribs or the sternum adjoining it. It may be soft or somewhat rough in character. It is produced in the pulmonary artery, probably from some interference with the lumen of the vessel near its origin. The precise way in which the lumen is affected is discussed elsewhere.[1] The murmur is often only audible when the patient is lying down, and usually disappears during a moderately deep inspiration. It is frequently present in cases of debility when there is no marked evidence of anæmia. It is followed by an accentuated pulmonary second sound.

B. *Tricuspid Murmur.*—The development of this murmur can under favourable circumstances be watched. In such cases we have found that it is audible first in the third and fourth left spaces near the edge of the sternum. When fully developed it is heard over the whole right heart,—that is, in the third, fourth, and fifth left spaces, extending further to the left the lower the interspace, but not reaching further out than a point about one inch to the inside of the apex of the left ventricle; over the sternum adjoining these spaces; and, in some cases, in the second, third, and fourth right spaces for an inch to one inch and a half from the edge of the sternum. This, as is seen in the frontispiece, is the part of the chest overlying the right heart. If the murmur be heard in the above positions when the patient is in the upright or sitting position, it may often be noted that, shortly after the recumbent posture is assumed, the murmur may in addition be heard over the manubrium sterni. The presence of the murmur in this latter situation has long been regarded as evidence that there was an aortic systolic murmur present. This, however, we consider to be a mistake,—the true explanation being, that in the recumbent position the distention of the right chambers becomes greater, and, owing to this distention and a greater reflux through the tricuspid orifice, the murmur is heard over a larger area. That increased distention occurs is shown by pulsation of the right ventricle and by increased fulness of the veins in the neck.

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[1] *Investigations into some Morbid Cardiac Conditions*, by William Russell, M.D. Edinburgh, 1886.

The murmur is due to reflux through the tricuspid orifice from the dilated and enfeebled condition of the right ventricle; and when it is present the pulmonary second sound is not accentuated.

C. *Mitral Murmur.*—This is the least common of the murmurs present in the conditions under consideration. It is, however, not infrequent, and is a soft blowing murmur, audible at the apex and propagated slightly outwards.

All these murmurs disappear as the patient recovers, and as the heart regains tone and strength.

Examination of the Blood-Vessels.

A. *Arteries.*—(1.) *Arch of Aorta.*—It is frequently necessary to investigate the condition of the arch of the aorta, as it, more than any other part of the vessel, is liable to aneurism. It is investigated by inspection, palpation, percussion, and auscultation.

Inspection may reveal pulsation above the base of the heart on either side of the sternum; more frequently, however, to the right than to the left of it. Pulsation may also be present in the episternal notch. In cases of large aneurism the sternum itself may move with the systolic expansion of the sac. In the most severe cases there is great bulging of the chest wall covering the aneurism, the bones become eroded, and there is thinning of all the structures overlying it, so that the sac ultimately may be covered only by integument.

By *palpation* the pulsation is felt, and its distensile character is, in marked cases, very distinct. In some cases a systolic thrill is present. The shock of the closure of the semilunar valves may also be intensified. When the aneurism is large and superficial, it must be palpated with great care. Caution is still more necessary when percussion is attempted.

Percussion is necessary in all save the most evident cases. By means of it we determine whether the percussion sound is modified over the manubrium. In aneurism and in other mediastinal tumours the percussion sound in this situation may be dull, the dulness extending to the right or left of the sternum.

On *auscultation* there may be a systolic murmur, but in a considerable proportion of cases there is no murmur. There is usually marked accentuation of the aortic second sound.

In addition to examining the aorta anteriorly it is necessary to *examine it posteriorly:* this is done in the interscapular region. It will be remembered that the aorta reaches the spine at the level of the third dorsal vertebra, so that a considerable aneurism, involving this part of the vessel, may give evidence of its presence by dulness on either side of the vertebral column. When the bodies of the vertebræ are eroded by pressure there is tenderness on pressure, and there may be even curvature.

The typical signs of the presence of aneurism are thus—pulsation, thrill, dulness on percussion, systolic murmur, and an accentuated second sound. There are, however, other evidences of its presence which have to be examined for. They are known collectively as *pressure signs*. Thus there may be evidence of—(1.) *Pressure on one bronchus*, as indicated by snoring rhonchi over the seat of pressure; (2.) *Pressure on the large venous trunks*, leading to fulness of the veins on one or both sides of the face, neck, and upper extremity, and even to œdema of these parts; (3.) *Pressure on the trachea* giving rise by its irritation to a peculiar brassy and hollow cough, or interfering with the extrance of air by direct pressure, and so producing an inspiratory stridor; (4.) *Pressure on the œsophagus*, producing difficulty in swallowing; (5.) *Pressure on the recurrent laryngeal nerve* on the left side where it turns up under the aortic arch, leading to paralysis of the left vocal chord; (6.) *Pressure on the sympathetic*, causing inequality in the pupils; (7.) *Pressure on the vagus or the phrenic*; (8.) *Pressure on bone*, as already referred to, causing caries and absorption. Finally, *the pulses* on the two sides have to be compared. In aneurism there is not infrequently a marked inequality, owing to the involvement of the origin of one of the large trunks arising from the arch in such a way that its lumen is diminished, and the circulation through it consequently impeded.

While all these evidences of aneurism have to be borne in mind, too much stress must not be laid on any one of them, for here, more than in many conditions, the presence of some symptoms, and the absence of others, have to be carefully weighed.

Atheroma of the arch of the aorta, with moderate dilatation, associated with hypertrophy of the heart and calcareous deposit at the bases of the aortic cusps, may be very readily, and is often, mistaken for aneurism. The differential diagnosis is probably often impossible, but the correspondence in the physical signs in the two conditions must be borne in mind. The points in which they correspond are the presence of pulsation in the episternal notch, increased area of dulness, systolic murmur, and accentuated aortic second sound. Pressure symptoms are not, however, present.

(2.) *Descending Thoracic Aorta.*—This part also of the vessel may be the seat of aneurism. If it is large its position may be made out by percussion, and there may be a murmur. If erosion of the vertebræ has supervened there is tenderness on pressure, while in severe cases there may be spinal curvature and paraplegia from implication of the cord. Minor degrees are hardly discoverable by physical examination.

(3.) *Innominate Artery.*—This vessel is often involved in aneurism of the aortic arch; it may, however, be affected apart from it. The bulging, unless the sac be very large, is to the right of the sternum, and reaches to the level of the sterno-clavicular articulation. The orifices of the right subclavian and aortic arteries are very liable to be implicated, and to lead to a difference in the radial and carotid pulses on the two sides. The other signs correspond with those of aneurism of the arch, but the pressure phenomena are not so varied.

(4.) *Abdominal Aorta.*—Aneurism of this part of the vessel will be found under the section on the abdomen.

(5.) *Carotid Arteries.*—Normally the pulse in these vessels is not visible. In double aortic lesion—that is, when there is both stenosis and regurgitation—the pulsation of the arteries, especially the right one, is visible right up to the ear. This is the only valvular lesion in which this phenomenon is present; it may, however, be present in exophthalmic goître without lesion at the aortic valves. We have already mentioned that systolic aortic murmur is propagated into and can be heard over the carotids. In addition to this, however, in cases of aortic regurgitation, if the stethoscope be pretty firmly applied to the carotid, *a diastolic murmur* can be heard. This murmur is not, of course, the diastolic murmur propagated from the orifice of the aorta, for murmurs are not propagated in a direction opposite to the direction of the stream producing them, but it is caused by the backward flowing stream in the vessel passing the part narrowed by the pressure of the stethoscope. This diastolic murmur can often be beautifully heard in the femoral arteries by carefully graduating the pressure with the stethoscope.

(6.) *Radial Arteries.—Examination of the Radial Pulse.*—From what has already been pointed out, it is clear that a study of the condition of the arterial pulse is fraught with disease indications of a far-reaching character; and in order to acquire skill, not only in the diagnosis of circulatory disorders, but in the investigation of all affections which have even a remote influence upon the circulatory system, a knowledge of the changes which it undergoes is in the highest degree necessary. Such knowledge can only be attained by diligent practice and patient study, since the modifications of the arterial pulse are so numerous and so various as to require much careful observation for their detection, while the student must undergo considerable training before he can possess the requisite faculty of appreciating the differences between the pulse of health and disease.

In the following pages the methods of ascertaining the state of the pulse will in the first place be considered, after which the changes which are to be found in different conditions will be described.

Method of observing the Pulse.—In this place attention must be called to the locality where the pulse may be most conveniently studied, and the means by which its investigation is to be carried out will afterwards be described.

The examination of the arterial pulse is usually confined to the radial arteries, and the part of the vessels subjected to investigation is that which lies immediately above the wrist, between the flexor tendons on the inner, and the prominent ridge of the radius on the outer side. In this situation the arteries are only covered by the skin and subcutaneous tissues; they can therefore be felt quite easily, and may often be seen as well. At this point, moreover, the vessel is in contact with the radius, and, in consequence of this fact, its examination is rendered at once more simple and more thorough, inasmuch as the bony floor upon which it lies gives a fixed base against which it may be firmly pressed.

The pulse should if possible be examined while the patient is in a sitting or lying posture, and the arm which is employed for the purpose must rest upon some fixed object, or be supported by the hand of the observer which is not made use of in palpating the artery. The observer should place himself in front of or to the right of the patient, whose right arm is to be in the semi-prone position, with the back of the hand turned to him; he must then pass his right hand across the radial surface of the patient's wrist, and lay the tips of his middle and ring fingers upon the vessel. If the patient's left radial artery is to be examined, the observer should stand to the left and employ his left hand in a similar manner; while if both radial arteries are to be examined simultaneously, he should as far as possible stand in front of the patient and use his right hand for the left artery and his left for the right. Attention to these details may seem unnecessary, but the adoption of this method will be found to render the examination of the pulse at the same time more easy and more precise. Laying the tips of the fingers at first lightly upon the artery, the pressure should be varied in order to bring out the different points, which will immediately be described.

The only instrument to which reference need be made as of any use in a study of the pulse is the sphygmograph, and, as it is nowadays so well known, a description would be as superfluous in its case, as in the case of the stethoscope. There are many modifications of the sphygmograph, but the only essential difference between them is that some have the pressure applied, as in Marey's instrument, by means of a spring, while others, such as Sommerbrodt's sphygmograph, have actual weights which bear upon the artery. Of the many varieties which are employed, the only ones which we are able to recommend are Sommerbrodt's, Marey's, and Mahomed's.

It must be borne in mind that the sphygmograph is of limited use in diagnosis. It reveals no new fact which can enable us to discover affections unknown before its invention; it nevertheless brings into prominence certain points in regard to the pulse which would be less definite without its aid, and it is on this account of some clinical interest. On the other hand, there are some aspects of the pulse which can be much more accurately gauged by means of the finger than by the help of the sphygmograph. The instrument can therefore only be regarded as a supplement to the finger of the observer.

In employing the sphygmograph there are a few points which must be attended to. The patient must, in the first place, assume a comfortable attitude whether sitting or lying, while the arm, the artery of which is to be examined, must be put in an easy position, almost fully supinated, and resting upon a steady support. The button of the instrument is then to be accurately placed upon the most prominent part of the artery, and, after the point yielding the largest movement of the lever has been ascertained, the sphygmograph is to be lashed to the wrist. The pressure, lastly, has to be adjusted, and it will be found that there is one definite amount which gives the fullest amplitude of movement, an increase or decrease of the pressure yielding a less perfect tracing. Such are the points which must be borne in mind. The use of the sphygmograph can only be acquired by dint of assiduous practice. In the following pages its indications will be mentioned along with the descriptions given of the different elements of the pulse, and their variation as ascertained by means of the finger.

It is necessary to recognise the fact that tracings taken from the same artery at the same time, but by means of different forms of sphymograph, may present very varied appearances. The two tracings which follow, for example, were obtained at the same time from the right radial artery of a patient suffering from mitral incompetence with hypertrophy of the left ventricle. The pressure in both cases was the same, but in the case of the tracing taken with Marey's instrument it was due to a spring, while in the case of that

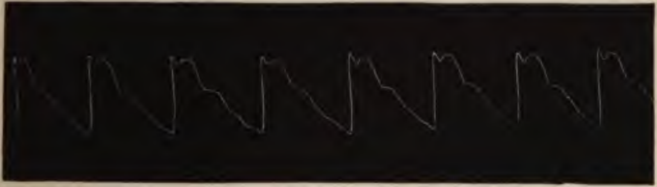


FIG. 27.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral incompetence; pressure 2 oz.

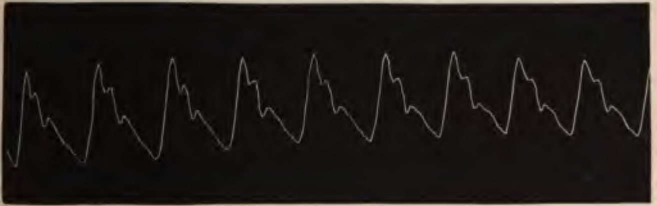


FIG. 82.—Tracing taken with Sommerbrodt's sphymograph from the radial artery in a case of mitral incompetence; pressure 2 oz.

obtained by means of Sommerbrodt's instrument it was applied by direct weight. It will be observed that the lever in the former tracing describes the arc of a circle from swinging parallel to the moving paper; while in the latter the lever gives a perfectly vertical line, because the paper travels at right angles to it. This gives the upstroke an absolutely true relation to the rest of the tracing. In tracings taken with Marey's and Mahomed's instruments the upstroke has always a backward inclination on account of the circular movement of the lever.

Phenomena to be studied.—In the observation of the characters of the pulse, it is convenient to separate out its different elements, and estimate each of them in turn. Several of these are doubtless only different phases of the same conditions, but it conduces to the ease of its investigation when the phenomena which the pulse presents are severally analysed. Attention should therefore be devoted in turn to the following points :—

1. The condition of the arterial walls.
2. The state of the blood supply, as regards—
    - *a.* The fulness of the vessel, and
    - *b.* The tension of the artery.
3. The character of the pulsation as regards—
    - *a.* The rate of the arterial pulse ;
    - *b.* The rhythm of the arterial pulse.
4. The character of each pulse-wave as regards—
    - *a.* The size of the wave ;
    - *b.* The force of the wave ;
    - *c.* The duration of the wave.
5. The character of the pulsation in different arteries in the same individual.

Normal Characters of the Radial Pulse.—The vessel should be soft and yielding, moderately filled with blood, and easily compressible. The rate of pulsation may vary greatly within certain limits, but the rhythm should be perfectly regular. Each pulsation should be of moderate and uniform size and force, while neither too long nor too short in duration.

A sphygmographic tracing of the pulse in health, as in Fig. 29, presents a sudden and uninterrupted line of ascent, and a gradual line of descent, marked by two distinct undulations. The line of ascent (*a-b* in Fig. 30) is almost vertical when taken with sphygmographs writing with a lever at right angles to the paper, and it inclines somewhat backwards when obtained with such a sphygmograph as Marey's. This line of ascent is commonly called the *percussion wave*, and it is caused by a swift wave of distention

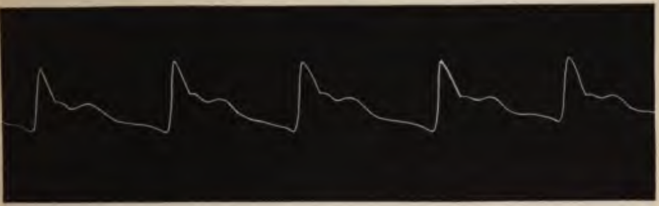


FIG. 29.—Tracing from pulse of healthy man; pressure 2½ oz.

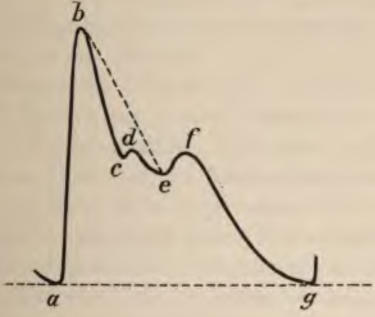


FIG. 30.—Diagram of pulse tracing in health.

produced by the sudden entrance of the blood from the left ventricle into the arterial system. It terminates in a sharp apex. The line of descent (*b-g* in Fig. 30) is caused by the more gradual contraction of the vessel after the cessation of cardiac systole. As above mentioned, it is interrupted by two distinct elevations. The first or higher of these (*d* in Fig. 30) is commonly known as the *predicrotic* or *tidal-wave*, and it is believed to be caused by the blood-stream coursing through the artery. The second or lower elevation (*f* in Fig. 30) is generally termed the *dicrotic* or *recoil-wave*, and it is attributed to the reflection of an impulse from the closed aortic valves. These waves vary, as will be seen below, with the blood-pressure, and it may be stated that their position depends on the distance between the heart and the artery from which the tracing has been obtained; the nearer the artery is to the heart, the higher upon the line of descent are these elevations.

1. *The condition of the Arterial Walls.*—The walls of the radial artery in health are soft and yielding, but they give at the same time a feeling of elasticity to the fingers which are applied to them. The state of these walls gives a reliable guide to the condition of the arterial system. The chief departure from this healthy state is hardness and rigidity of the vessel, caused by thickening or atheromatous degeneration. The artery may be so hard and resistent as to roll from side to side under the finger. Such a condition is common in advanced years, and may be taken as indicating a tendency to cerebral or renal mischief. In order to discover if there is any tortuosity of the vessel, the observer should pass his fingers along its course up the arm.

Care must be taken to distinguish this condition from the state of the circulation known as high tension, caused by increased blood pressure. They are frequently associated together, and the means of differentiating the two conditions will be mentioned.

Tracings taken by the sphygmograph from patients whose arteries are atheromatous show a low upstroke, with a blunted apex, and very little tendency to any oscillations during the descent of the lever. The limited range of movement during the ascent of the lever, and the blunt appearance of the summit, are due to the rigidity of the walls, which also causes the absence of the usual waves seen on the descending line of the tracing.

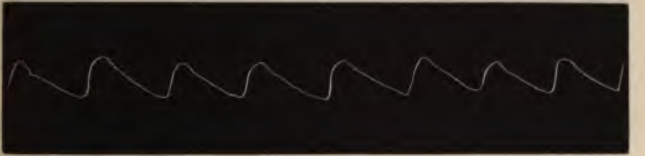


FIG. 31.—Tracing taken from a case of advanced atheroma of the arteries by means of Marey's sphygmograph; pressure 3 oz.

2. *The state of the blood supply* may be estimated by ascertaining the fulness of the artery, and the tension or degree of blood pressure.

(*a.*) *The fulness of the vessel* depends on several factors, the principal of which are—the quantity of blood in the circulation, the energy of the cardiac contractions, and the calibre of the arterioles, caused by the activity of the vaso-motor nervous system. The fulness of the vessel is to be judged by its size during the interval between two pulsations, and the sphygmograph renders no help in the determination of this point. As a rule, a full pulse (*pulsus plenus*) is associated with a fairly large pulse-wave; but it is very common to find that the pulse-wave may be very small although the vessel is full. An empty pulse (*pulsus vacuus*) is, on the other hand, very frequently found along with large and bounding pulsations, as in the typical Corrigan's pulse of aortic incompetence.

A full pulse is common in persons of a plethoric habit, and in most cases of increased tonicity of the small arteries, such as the early stages of chronic granular kidney. An empty pulse is found in malnutrition and wasting diseases, as well as the stage of convalescence in acute affections. It is further to be observed in conditions of relaxation of the small arteries and in incompetence of the aortic valves.

(*b.*) *The tension of the artery* is intimately associated with the condition of the vaso-motor system, but it has relations also with the activity of the heart and the quantity of blood poured into the arterial system during the cardiac systole.

It is to be estimated by the amount of force necessary to obliterate the artery during the interval between the pulsations, and the common term *compressible* is synonymous with a pulse of *low tension*, while *incompressible* is a term equivalent to a pulse of *high* tension. It may also be gauged by the amount of expansion which the artery undergoes during the cardiac systole; a pulse of high tension is, *cæteris paribus*, one with a smaller pulse-wave than a pulse of low tension.

Care must be taken not to mistake atheroma of the arterial walls (see page 96) for increased arterial tension.

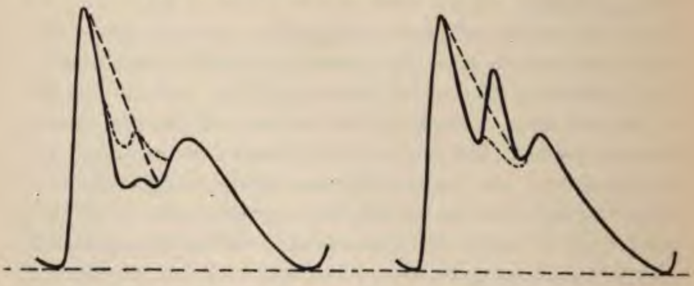


FIG. 32.—Diagram of pulse of low tension.

FIG. 33.—Diagram of pulse of high tension.

The finely dotted line represents the normal curve.

Sphygmographic tracings give a reliable means of estimating the tension of the artery and pressure of the blood. If a line be drawn from the apex of the tracing to the lowest point of the aortic notch, as in the following figures, it will be found that the tidal wave is sometimes below it, and at other times reaches or passes it. If it is below it, as in Fig. 32, the pulse is of low or moderate tension, but if, as in Fig. 33, it reaches as high as, or higher than, the line, the pulse is of high tension.

For the most part the pulse-wave is small in cases of high tension, but this is not invariably to be found. In Bright's disease the pulse-wave may be large, in peritonitis it is small; both conditions have high tension.

With low tension the pulse-wave may be large or small. In the acute stage of febrile affections, and in aortic incompetence, it is usually large; in the later stages of febrile diseases, and in mitral stenosis, it is as a rule small.

In connection with the subject of arterial tension, it is natural to consider what is known as *dicrotism*. When a healthy vessel is palpated, only one pulsation can be felt

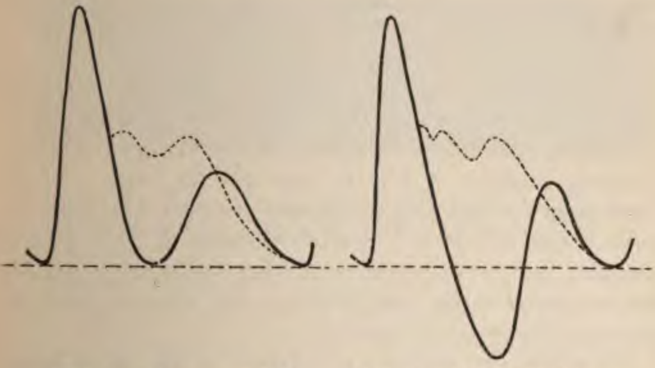


FIG. 34.—Diagram of fully dicrotic pulse.

FIG. 35.—Diagram of hyper-dicrotic pulse.

The finely dotted line represents the normal curve.

corresponding to each contraction of the heart, but in certain conditions a second wave can be felt immediately following it. A tracing of such a pulse shows a greater or less exaggeration of the recoil-wave, and an almost or quite absolute disappearance of the tidal wave. When the notch preceding the wave of recoil reaches the base line, as in Fig. 34, the pulse is said to be *fully dicrotic*, and when this notch sinks below the base line, as in Fig. 35, the term *hyperdicrotic* is applied to it.

It need hardly be added that dicrotism and hyperdicrotism are invariably results of low tension.

3. *The character of the pulsation* must be observed with regard to its rate and rhythm.

(*a.*) *The rate of the pulse*, or the number of pulsations in a given interval of time, varies with changes in cardiac activity, blood supply, and arterial tone.

In an adult man the number of pulsations usually varies between 60 and 70 in a minute, but there are personal idiosyncrasies which in conditions of apparent health may lead to the rate of pulsation being as low as 20 or as high as 100 per minute. An increase in the rate constitutes a *frequent* pulse (*pulsus frequens*), and a diminution in the rate is termed an *infrequent* pulse (*pulsus rarus*).

The rate varies in health with sex, age, size, position, external temperature, hour of day, condition of digestion, occupation, and many other less important circumstances. Speaking generally, it may be said that the pulse is more frequent in the following conditions:—in the female sex; in infancy and childhood; in small persons; in the upright attitude; in high temperatures; during the later periods of the day; after eating and drinking; and when engaged in any mental or muscular exertion.

The rate of the pulsation is increased by the febrile state, and the increase is in almost all diseases directly proportional to the rise of temperature. In enteric fever and cerebral inflammations this direct ratio is usually absent. The pulse is increased in frequency in certain nervous diseases, especially exophthalmic goître, and nervous palpitation. It is also more frequent in almost all the organic diseases of the heart, the only common exception being aortic stenosis.

The pulse is rendered less frequent in the critical stages of fevers, especially when there is a tendency to collapse; in certain disorders of the blood, such as jaundice; in high tension pulses,—as, for example, in renal affections; in inflammations and degenerations of the muscular structure of the heart; and in some affections of the membranes of the brain.

(*b.*) *The rhythm of the pulse* is absolutely regular in health, but there are personal idiosyncrasies which lead to irregularity without any apparent cause.

The rhythm depends entirely upon the nervous influences governing the motor impulses giving rise to the cardiac systole, and these nervous influences may be disturbed by alterations in blood pressure, such as want of compensation. In mitral stenosis the irregularity of the pulse is a marked feature of the disease even at an early stage, as in Fig. 36.

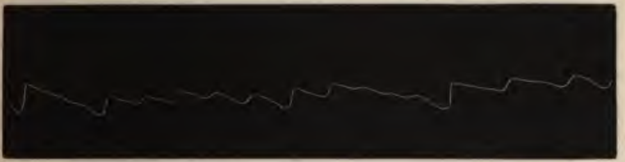


FIG. 36.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral stenosis; pressure $2\frac{1}{2}$ oz.

Irregularity of the pulse is frequently found in cardiac degeneration and in nervous diseases, and it is quite a common symptom in elderly persons without any apparent disease.

Irregularity of the pulse frequently assumes more or less periodicity in its occurrence,—that is to say, the irregularity recurs at regular intervals.

The omission of pulse-waves constitutes the symptom known as *intermission* (*pulsus deficiens*), while the intercaladistinguished from the fulness of the vessels which has already been discussed.

(*b*.) The *force* of each wave is closely related to its size, but volume and strength are not invariably associated. The bounding pulse of arterial relaxation is very often feeble. The force depends in the main upon the energy of the cardiac systole, but to some extent also on the degree of tone possessed by the arterioles.

In all conditions of increased cardiac activity, the force of the pulse is increased, while in debility of the heart it is diminished.

(*c*.) The *duration* of each pulsation depends upon the relation existing between the dilatation of the artery by the blood-current, and its contraction during the passage of the blood into the capillaries. The duration is longer when the blood has considerable opposition to overcome in passing out of the arteries, and shorter under converse circumstances. It is also longer when the systole of the heart is prolonged, and shorter when the cardiac contraction is brief.

As tested by the finger, the quick is readily distinguishable from the slow pulse by the duration of the impact felt by the observer. The sphygmographic tracing of the two shows a quicker ascent, sharper apex, and swifter descent in the former than in the latter.

A *slow* or sluggish pulse (*pulsus tardus*) is almost invariably associated with high arterial tension; but this is not an absolute rule, as in aortic stenosis, for example, where there is much obstruction, the pulse may be extremely sluggish, although the tension is low. A slow pulse is found in all affections where there is contraction of the smaller arteries, such as angina pectoris, renal disease, and arterial sclerosis.

A *quick* or active pulse (*pulsus celer*) is found, as a rule, along with low arterial tension, and it depends upon a relaxed condition of the arterioles for the most part. It is extremely common therefore in febrile affections. A very marked variety of the quick pulse is to be observed in cases of free aortic regurgitation, where the celerity is associated with an empty condition of the artery, and a large pulse-wave.

In health the individual pulsations should all be equal in size, force, and duration; but it is found that when there are irregularities in rhythm, there are almost invariably inequalities in size and force.

5. *The characters of the pulse in different arteries in the same person* may require attention, in order to determine the presence of certain local interferences with the current through the arterial system. In similar arteries, on different sides of the body, the pulse under ordinary circumstances occurs at the same instant of time, and if there is a difference in time between the pulsation on the two sides, some obstacle is the cause of the delay in the later pulse. In the same way there is a definite interval of time between the pulse in arteries situated at different distances from the heart, between the carotid and radial for instance, or between the radial and femoral or tibial. If the usual relation between the pulse in such vessels should be altered, some obstacle is here again the cause of the change.

When there is any delay in the pulse of a vessel, there is usually at the same time some change in the characters of the wave. In aneurism of the arch of the aorta, for example, there may be a considerable difference in time and volume between the pulse of the right and left radial arteries.

The common causes of such local variations are abnormal distribution, or obliteration of vessels, aneurisms, thrombosis, embolism, pressure of tumours, and injuries involving the course of the artery.

B. *Venous Capillaries.*—The distention of these produces the condition known as cyanosis.

C. *Arterial Capillaries.*—A very interesting phenomenon is developed in the arterial capillaries in marked cases of aortic regurgitation. If the nails be examined, an alternate flushing and pallor may be seen. The phenomenon can also be produced by drawing the finger nail across the forehead of the patient so as to produce a red line, when its alternate flushing and pallor is perceptible. This has been described as the *capillary pulse.*

Inspection of the Veins of the Neck.—The veins of the neck present extremely different appearances in stout and in thin people. In the former they can only be seen with difficulty; in the latter they stand out distinctly both in form and colour. It is necessary to pay attention to the condition of the jugular veins in regard to *fulness* and to *movements.*

The *fulness* of the jugular veins may be increased by such causes as thrombosis of one of the great veins nearer the heart, or pressure upon such a vessel by a tumour. In such a case the venous engorgement is for the most part unilateral. The fulness may on the other hand be augmented by such causes as the various dilatations and valvular lesions of the heart, and many chronic lung affections. In cases of this kind the venous stasis affects both sides alike, and is but part of a disturbance of the circulation in general.

The *movements* of the veins depend upon two groups of causes—*respiratory* and *circulatory conditions.*

Movements caused by Respiration.—In thin persons undulations along the jugular veins, synchronous with the movement of respiration, may often be seen. These undulations consist in a gradual filling during expiration, and a sudden emptying of the veins at the beginning of inspiration. The explanation is obvious: the fulness during expiration is the result of the positive pressure on the veins of the thorax during that phase, which causes a retrograde wave of blood to close the valve above the jugular bulb; the sudden emptying attending inspiration is produced by the negative pressure within the thorax at that time, which draws the blood onwards in the direction of the heart.

Movements caused by Circulatory Conditions.—In addition to these movements caused by respiratory conditions there are others solely connected with the circulatory functions. These are grouped together under the term *venous pulse.*

The venous pulse *in health* consist of slight undulations which are invariably earlier in time than the apex-beat; the venous pulse is therefore, to use the common phrase, presystolic. This pulsation is only seen in very thin persons, and it is, as a rule, more visible in the external than in the internal jugular vein. It is produced by the contraction of the walls of the great veins and right auricle, which causes a current to flow backwards as far as the valves on the veins. The blood current is stopped there, but the closure of the jugular valve communicates a distinct pulsation to the contents of the vein beyond, and every separate shock received by the valve is similarly transmitted to the blood above.

Tracings obtained from the external jugular vein show at times a single wave, cause by auricular systole. There may, however, be two impulses, in which case the second wave is produced by a shock transmitted backwards from the heart, during the closure of the auriculo-ventricular valves. At rare times there may even be a third wave, agreeing in time with the diastole of the heart, and no doubt communicated to the veins by the shock caused by the closure of the arterial valves.

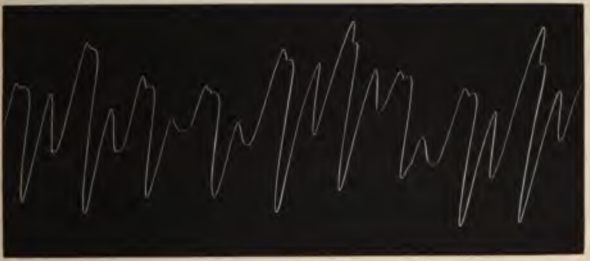


FIG. 39.—Tracing from the internal jugular vein in a case of tricuspid incompetence.

The venous pulse in *diseased conditions* is essentially different. This pulse usually consists of two distinct waves, the first and smaller being produced by the systole of the right auricle; the second and larger being caused by a wave of regurgitation from the ventricle into the auricle. This form of pulsation of the jugular veins is pathognomonic of tricuspid incompetence. A tracing of such a venous pulse is annexed, in which the auricular and ventricular waves can be clearly seen. The exact form of these waves varies greatly, and the tracings obtained from the internal and external jugular veins differ greatly in details, although their main curves are similar.

Friedreich has pointed out that in some cases paralysis of the auricle can be detected by the absence of the auricular wave, which he states to have disappeared in the course of cases under his care.

THE BLOOD.

In many cases the blood requires to be examined to ascertain the following points:—its richness in red corpuscles, their size and shape, and the amount of hæmoglobin they contain; the number of white corpuscles, and the proportion they bear to the red; and the presence of microparasites.

Red Corpuscles.

Diminution in the number of Red Corpuscles or Oligocythæmia occurs in anæmia, from whatever cause. In the normal condition the blood contains about five millions of red corpuscles in each cubic millimetre, while in different abnormal states the degree of diminution varies of course in proportion to the severity of the condition. In chlorosis, for instance, they may fall to two and a half or three millions, while in pernicious anæmia they often fall to about half a million before death. Their number is usually estimated in this country by means of Gowers' *Hæmacytometer*. This instrument consists of — *A*, a small pipette, marked to contain 995 cubic millimetres; *B*, a capillary tube, marked to contain 5 cubic millimetres (both these are fitted with india-rubber tubes and mouth-pieces to facilitate their being accurately filled); *D*, a small glass vessel to contain the blood and diluting fluid; *E*, a glass stirrer; *C*, a brass stage-plate, carrying a glass slide, in which is a cell one-fifth of a millimetre deep, and the bottom of which is divided by intersecting lines into squares of one-tenth of a millimetre, while a cover-glass is retained in position over the cell by means of two clips; and *F*, a guarded needle to pierce the finger with.

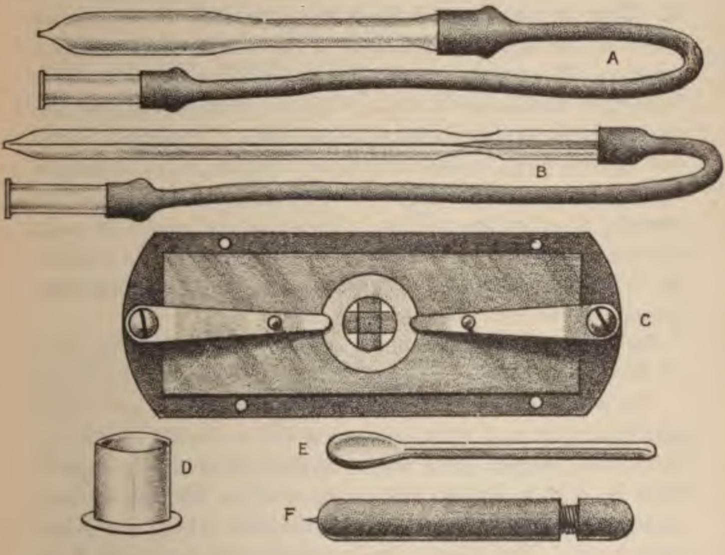


Fig. 40.—Gowers' Hæmacytometer. *A*. Pipette; *B*. Capillary tube; *C*. Stage-plate; *D*. Glass vessel; *E*. Stirrer; *F*. Guarded needle.

When an enumeration is to be made, 995 cubic millimetres, measured by means of the pipette *A*, of a solution of twenty-six grains of sulphate of soda to an ounce of distilled water, to which has been added fifteen drops of strong acetic acid, are put into the glass vessel *D*, and to this is added five cubic millimetres of blood drawn from the finger by the needle *F*. These are well stirred together, and then a drop is put in the centre of the cell, and the cover-glass put over it. This is placed under the microscope, and the corpuscles in ten of the squares, as marked on the bottom of the cell, are counted. This number, multiplied by 10,000, gives the number in a cubic millimetre of blood. The corpuscular richness can also be expressed as so many *per centum*. In normal blood two squares contain one hundred red corpuscles, so if twenty squares are counted and added together, and then divided by twenty, it gives the average number of corpuscles to each square, and if this be multiplied by two, it gives the number in two squares, and that is the per-centage of corpuscles present in the case under examination.

By examination of the blood at short intervals the progress of the case and the effect of remedies can be closely followed.

The size and shape of the Red Corpuscles.—In pernicious and other forms of chronic anæmia the red cells vary much in size and shape. In all forms the *average* size is diminished. This in part is due to the presence of a number of very small and very deeply coloured corpuscles, which are called *microcytes*. The blood also contains some unusually large corpuscles, known as *megalocytes* or *macrocytes*. The corpuscles also vary in shape, becoming pear and club shaped, and assuming various other irregular forms—the term *poikilocytosis* indicating these variations in shape.

Variations in the amount of Hæmoglobin.—The amount of hæmoglobin in the blood may be lowered absolutely and relatively; for instance, both in chlorosis and pernicious anæmia the amount of hæmoglobin is absolutely diminished, but in the former the diminution is relatively greater owing to the exceeding poorness of the individual corpuscles in hæmoglobin, while in pernicious anæmia the absolute diminution is greater than in chlorosis, while relatively, instead of diminution, there is increase owing to the increased richness of the individual corpuscles.

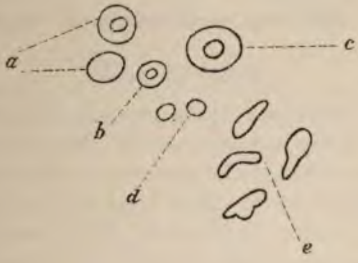


FIG. 41.—Forms of blood corpuscles in anæmia (after Scheube). *a*, normal ; *b*, small red corpuscles ; *c*, macrocytes ; *d*, microcytes ; *e*, irregular forms of red discs.

The estimation of the hæmoglobin may be made by Gowers' *hæmoglobinometer*. The apparatus consists of a capillary pipette and two glass tubes, one of which contains a standard colour solution, while the other is graduated so that 100 degrees equals two cubic centimetres. The method is as follows :—Some drops of distilled water are put into the graduated tube, and to this is added 20 cubic millimetres of the blood to be examined, the quantity being measured in the pipette, and the two are quickly shaken together. Distilled water is then added drop by drop until the diluted blood is of the same shade as the standard colour solution, and the degree at which it stands is the per-centage of hæmoglobin present, *i.e.*, if the diluted blood gives the standard colour at 60, the per-centage of hæmoglobin is 60.

The average richness of the individual corpuscles is expressed by a fraction, the numerator of which is the percentage of hæmoglobin as found above, and the denominator the per-centage of corpuscles as found by the hæmacytometer. Thus if in a case of chlorosis the hæmoglobin richness is 40 per cent., and the corpuscles are reduced to 50 per cent. the average richness of the individual corpuscle is expressed by the fraction $\frac{40}{50}$ or $\frac{20}{25}$, and this would further show that the proportional deficiency of hæmoglobin is greater than of corpuscles.

White Corpuscles.

Normally there is one white corpuscle in about six squares, as reckoned by the hæmacytometer. Minor variations are of no clinical or pathological significance so far as is known. In leucocythæmia, however, there is an absolute increase in the number, and, owing to the fall at the same time in the number of red corpuscles, the white are relatively still more abundant, the proportion rising even to 1 in 20.

Microparasites.

These are animal or vegetable. Of the first the only one of importance is the *Filaria sanguinis hominis*, which appears in great abundance in the blood during the hours of sleep of the individual harbouring the mature worm.

The vegetable microparasites are numerous: among the more important may be mentioned the bacillus of anthrax, the spirillum of relapsing fever, and various bacilli and micrococci in septic and other fevers. These require special methods of staining for their demonstration, for which reference is necessary to works on practical pathology.

Spectroscopic Examination.

Both arterial and venous blood give a distinctive spectrum. The recognition of blood by the spectroscope will be referred to in the section dealing with the urine.

CHAPTER VI.

RESPIRATORY SYSTEM.

THORAX—SURFACE LINES AND AREAS.

FOR convenience in description, the thorax is regarded as presenting several *areas*, and marked by six *vertical lines*.

The lines are (*a*) the mammary line, a vertical line passing through the mamma; (*b*) the parasternal line, drawn midway between the preceding and the edge of the sternum; (*c*) the anterior axillary line, drawn downwards from the anterior

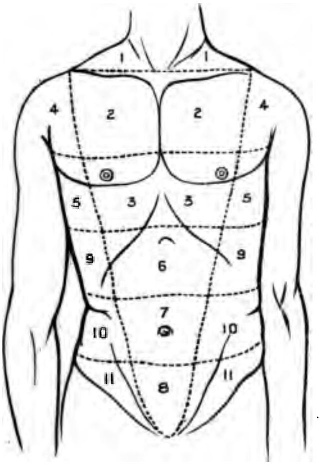


FIG. 42.—Anterior thoracic and abdominal regions.

1. Supra-clavicular; 2. Infra-clavicular; 3. Mammary; 4. Axillary; 5. Infra-axillary; 6. Epigastric; 7. Umbilical; 8. Hypogastric; 9. Hypochondriac (right and left); 10. Iliac (right and left); 11. Inguinal (right and left).

fold of the axilla; (*d*) the mid-axillary line, drawn from the centre of the axilla; (*e*) the posterior axillary line, from the posterior fold of the axilla; and (*f*) the scapular line, drawn through the angle of the scapula.

The areas are anteriorly (*a*) the supra-clavicular, above the clavicle; (*b*) the infra-clavicular, from the clavicle to the third rib; and (*c*) the mammary, from the third rib downwards.

Posteriorly they are (*a*) supra-scapular, above the scapula; (*b*) the scapular, which may be divided into a supra—and infra—spinous; (*c*) the inter-scapular; and (*d*) the infra-scapular.

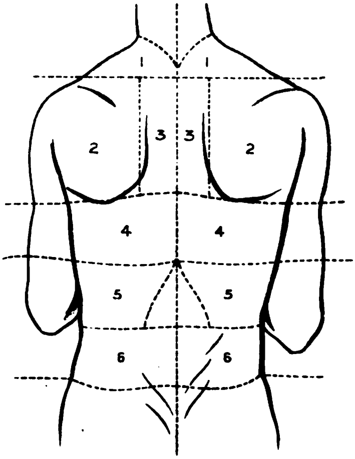


Fig. 43.—Posterior thoracic and abdominal regions.

1. Supra-scapular; 2. Scapular; 3. Inter-scapular; 4. Infra-scapular; 5. Inferior dorsal; 6. Lumbar.

Laterally they are the axillary and infra-axillary.

They are shown, along with the abdominal regions, in the above figures.

ANATOMICAL RELATIONS OF THE LUNGS.

The *apices* of the lungs rise *anteriorly* to an inch or an inch and a half above the clavicles; *posteriorly* they reach the level of the seventh cervical vertebra. The *base* or inferior limit of both lungs is at the level of the sixth rib in the mammary line, the eighth rib in the axillary line, the ninth rib in the scapular line, and the tenth rib close to the spine. The anterior margins of the lungs are, during inspiration, in contact under the sternum from the second to the fourth rib. At the level of the fourth rib the edge of the left lung turns outwards and downwards, forming a notch, with its concavity directed to the middle line; the inferior limit of the notch joins the inferior edge of the lung at the sixth rib about the mammary line. This notch leaves a considerable part of the anterior surface of the pericardium in immediate contact with the internal surface of the chest wall. The bases of the lungs rest on the diaphragm, and are somewhat concave to fit its dome-like upper surface, while their margins become wedge-shaped where they lie between the outer aspect of the diaphragm and the chest wall.

The right lung consists of three lobes, the left of two. The relations of the lobes to the chest wall are of importance clinically, and may be tabulated as follows :—

RIGHT LUNG, 3 lobes,	*Anteriorly* (*Mammary line*).	*Laterally.*	*Posteriorly.*
Upper lobe, . . .	From apex to fourth or fifth rib.	To fourth rib.	From apex to spine of scapula.
Middle lobe, . .	From fourth or fifth rib to inferior margin of lung.	From fourth to sixth rib.	*Nil.*
Lower lobe, . . .	*Nil.*	From sixth to eighth rib.	From spine of scapula to tenth rib.
LEFT LUNG, 2 lobes.			
Upper lobe, . . .	From apex to sixth rib.	To fourth rib.	From apex to spine of scapula.
Lower lobe, . . .	*Nil.*	From fourth rib to base.	From spine of scapula to base.

occurs in early life. In addition there is present a row of bead-like projections on each side, at the junction of the cartilages to their respective ribs, and known as the rachitic rosary.

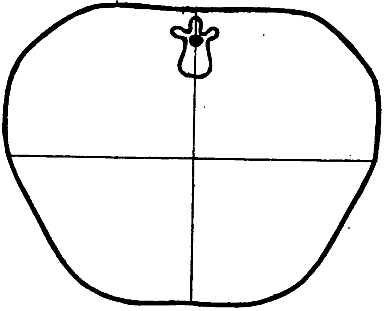


FIG. 44.—Normal chest (Gee).

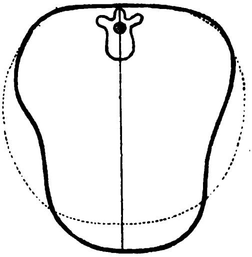


FIG. 45.—Rickety chest.
(Gee.)

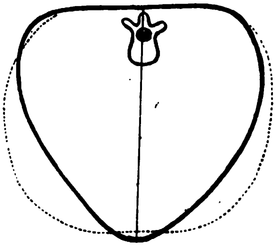


FIG. 46.—Pigeon-breast.
(Gee.)

B. *The pigeon-breast.*—This form is characterised by its triangular shape, the sides of the chest meeting almost at an angle. The sternum is in this way carried forward, and has been compared to the keel of a boat.

C. *The emphysematous.*—The chest becomes more barrel-shaped than normal in this form; that is, it becomes rounder, the transverse and antero-posterior diameters approaching each other in size, the former becoming smaller while the latter enlarges. The intercostal spaces are, as a rule, sunken. The sternum is arched forwards.

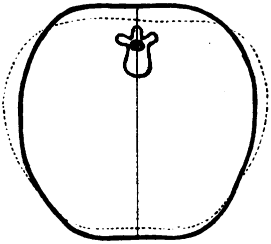


FIG. 47.—Emphysematous chest (Gee).

D. *The alar.*—This form is so named from the wing-like projection of the scapulæ. It is associated with a long narrow chest and greatly sloping shoulders. It occurs in persons of a phthisical inheritance.

Contraction of one-half of the thorax may follow on pleurisy or empyema. It is also present in fibroid phthisis and other destructive changes in the lung. The contraction is of course on the affected side.

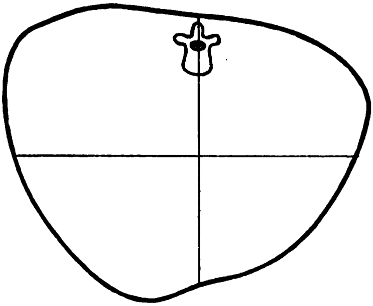


FIG. 48.—Showing retraction of one side of the chest (Gee).

Flattening under the clavicles occurs in cases where the apices of the lungs have been the seat of destructive changes, annulling or modifying their respiratory function. It is therefore associated with diminished respiratory movement. It may be present at both apices or confined to one.

In addition to the foregoing, which are those most commonly described, it is well to remember that in *angular and lateral curvature of the spine* the shape of the thorax is much altered, and that the limits and relative volume of the two lungs may be materially modified. In pronounced lateral curvature, owing to the altered curve of the ribs, and the altered volume of the lung, it will be found that the percussion sound is different on the two sides. Unsatisfactory physical signs must not in such cases have too much importance accorded to them if there is not other evidence of pulmonary disease.

B. *Size.*—The size of the chest in its circumference, and its vertical and horizontal diameters, varies greatly within the limits of health.

The following measurements are those usually made :—

The circumference is often the only measurement which is made. It is taken at the level of the nipples. The average is 34 inches, but it varies from 28 to 44 inches in the adult male. The chest expands from 1½ to 5 inches during forced inspiration, and it is well to observe this in health, as the diminution in the degree of expansion in various morbid conditions is to be specially noted. It occurs, for instance, to a very marked degree in emphysema and asthma, in both of which the measuring line may not indicate more than from a quarter to half an inch of increased circumference during inspiration.

The semi-circumference of the chest is greater on the right side than on the left by about half an inch. This measurement is taken from the spines of the vertebræ behind to the middle of the sternum in front : it is well to make a mark at both points before measuring. *Distention* of one-half occurs in pleuritic effusions when very copious, and also in some cases of pneumo-thorax. *Retraction* on one side occurs in fibroid induration of one lung, and after some cases of pleurisy or empyema.

In addition to the foregoing the *antero-posterior* and *transverse diameters* are of importance. They are taken by means of an ordinary steel *callipers.* Normally the transverse is greater than the antero-posterior diameter, but in abnormal conditions this may be considerably modified as has been seen in the preceding pages.

2.—*Movement.*

While in the normal state a more or less general expansion of the thorax occurs during inspiration. There are conditions in which this is modified or reversed. They may be classed under these heads :—

A. Diminished expansion—general or local.
B. Increased expansion—general or local.
C. Drawing in of the intercostal spaces—retraction.
D. Alterations in rhythm and rate.

A. *Diminished Expansion.*

Diminished expansion may be *general* and affect both lungs, as in emphysema, asthma, &c.; it may be *unilateral*, as in pleuritic effusion, pneumonia, &c. ; or it may be *local*, as at the apices from phthisis, elsewhere from pleurisy, collapse, &c. It occurs as a result of three main causes,—first, non-entrance or obstruction to the entrance of air into the lungs ; second, such a distended state of the pulmonary vesicles as will not admit of much more distention ; third, paralysis of the muscles which move the ribs.

First. Non-entrance, or obstruction to the entrance of air into the lungs. This condition may be the result of causes (1.) in the upper and larger respiratory passages ; (2.) in the smaller bronchi ; or (3.) in the pulmonary vesicles.

Under the first are included croup, paralysis of the vocal chords, larnygeal tumours, the pressure of tumours on the trachea or large bronchi, &c. Under the second—asthma; the obstruction being caused by spasm of the smaller bronchi. Under the third—phthisis, compression of the lung by pleural effusion, congenital or acquired collapse, pneumonic consolidation, and acute pleurisy, pleurodynia, or intercostal neuralgia. In the last of these the painful part is restrained owing to the increase of pain experienced on movement.

Second. An already distended condition of the pulmonary vesicles. This is typically seen in emphysema, the lungs and their containing case being so abnormally distended that even with deep inspiration the thorax may only expand about half an inch.

Third. Paralysis of the muscles of respiration which act on the thorax. This occurs in certain cerebral and spinal diseases or injuries, respiration being maintained by means of the diaphragm.

B. *Increased Expansion.*

General increased expansion occurs during violent exercise, and as a result of muscular effort. In the early stages of febrile affections there is, along with acceleration of the breathing, increased general expansile movement.

Local increase may affect one-half of the chest as when the lung on the opposite side is not performing its function. It is present when air or fluid in the opposite pleural cavity compresses the lung, also when there are destructive changes in the lung itself, and when the bronchus on one side is compressed or has its lumen obstructed in any way. When the apices of the lungs are the seat of tuberculous infiltration, the respiratory movement of the lower part is increased; and when the lower parts are infiltrated with pneumonic exudation, or compressed by fluid, the upper part shows increased movement.

C. *Drawing in, or Retraction.*

(*a.*) Drawing in of the intercostal spaces, the epigastrium, and the episternal and supra-clavicular regions, occurs during inspiration in cases in which there is obstruction to the entrance of air. It occurs from a variety of causes. The obstruction may be in the upper part of the respiratory passage, as in croup ; it may be lower down from the pressure of an enlarged thyroid, or of an aneurismal or other mediastinal tumour on the trachea or bronchi, or it may be due to spasm of the smaller bronchi, as in some cases of asthma or bronchitic asthma.

(*b.*) *Local drawing in* of the intercostal spaces occurs over areas of congenital or acquired collapse : this is frequently seen in children, and is most marked in the spaces between the asternal ribs anteriorly and laterally.

In all cases the explanation is that the capacity of the thorax is increased by the movement of its bony and cartilaginous framework, while at the same time the lung tissue does not expand : as a result the intercostal spaces and other yielding portions are driven in, for the atmospheric pressure remains constant while the pressure within the lung is necessarily diminished when the capacity of the thoracic cavity enlarges, and the entrance of air is obstructed.

D. *Alterations in Rhythm and Rate.*

Dyspnœa. — This term denotes increased rapidity or increased depth of respiration. It is brought on by physical exertion, but also by various morbid conditions. Elevated temperature is usually accompanied by accelerated respiration, while in other conditions there is not only acceleration, but increase in the depth of the respirations. When dyspnœa is caused by any hindrance to the free ingress or egress of air, it is known respectively as inspiratory or expiratory dyspnœa. *Inspiratory dyspnœa* is present in croup, the paroxysms of whooping-cough, paralysis of the vocal chords, or any condition in which the lumen of the upper part of the respiratory passages is diminished, as by the pressure of aneurismal and other tumours. *Expiratory dyspnœa* is present in the more advanced cases of bronchitis and emphysema, in which there is both obstruction in the bronchi and a diminution in the elasticity of the lung. It will be remembered that expiration depends on the elastic recoil of the lungs, and consequently, when this is weakened, expiration must be impaired.

Orthopnœa is a very marked degree of difficulty in breathing, the term being confined to cases where the patient requires to remain in the sitting or standing posture. It is present in the advanced stages of cardiac disease and in asthma, &c. In it the accessory muscles of respiration are called into action. The diseases mentioned above as giving rise to dyspnœa do in the more severe instances produce orthopnœa.

Cheyne-Stokes Respiration.—This is the best marked type of alteration in the respiratory rhythm. When fully developed it is characterised by a repeating cycle of phenomena; beginning with a number of superficial respirations it goes on to deep respirations, often amounting to dyspnœa, from which it falls again to superficial respirations resembling those with which it commenced; these are often called the ascending and descending phases, and following them is a period of complete cessation of the respiratory movements. The whole cycle occupies from a few seconds to two minutes, the pause occupying about one-half the whole time. The following tracing represents graphically the phenomena in this condition.

It may occur in all affections involving the cerebral and medullary centres. It is frequently present in uræmia and many acute diseases.

While the above is the fully and typically developed phenomena, there are many minor forms of irregularity which are allied to the fully developed form, but require no special description; some of these are not infrequently referred to as *cerebral breathing.*

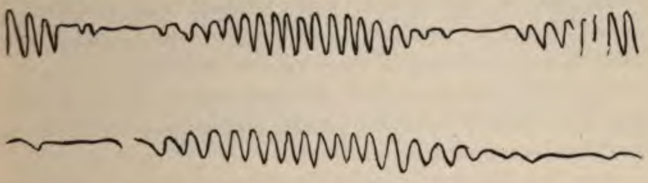


FIG. 49.—Tracings from cases of Cheyne-Stokes breathing.—(Gibson.)

The Respiratory Action of the Diaphragm.

This has been already referred to at the beginning of the chapter, but it is again necessary to draw attention to the importance of noting the extent to which respiration is performed by the diaphragm in different conditions. In emphysema, for instance, respiration is largely performed by it; while, on the other hand, if it is paralysed, or much impeded in its action by abdominal conditions, as ascites, &c., dyspnœa becomes extreme. No record of the respiratory functions which overlooks this can be regarded as complete.

II.—PALPATION AND VOCAL FREMITUS.

Palpation.—By palpation the observations made by inspection are verified and amplified. The sense of touch aids the sense of sight. The degree of movement is noted, and the student should familiarise himself especially with the expansile movement of normal respiration. The hands should be laid flat and firmly upon the corresponding parts on the two sides. Palpation of the apices anteriorly is more easily performed by standing behind the patient, when that is practicable, and putting the hands across the shoulders; in the same way, for palpation of the apices posteriorly, the hands may be put across the shoulders from the front. The lower part of the thorax, both laterally and posteriorly, should always be palpated as well as the apices, and a note made of the movement in these various situations.

The conditions in which deficient expansion is present have been already mentioned under inspection, and need not therefore be repeated here.

Pain on palpation is sometimes present, and may be even acute. It may be present either in the intercostal muscles, when it is usually rheumatic, or it may be confined to the nerves. It is also present before the appearance of the eruption in herpes zoster (shingles). In some cases of pleurisy pain is complained of on palpation, especially if considerable pressure be made on the part.

Vocal Fremitus.—The physical explanation of vocal fremitus has been already considered. The factors which determine its intensity may be classed as follows:—

(*a.*) The pitch of the voice: the lower the pitch the more marked the fremitus, and *vice versa.* This explains the fact that in women it is, as a rule, less marked than in men, the former having the higher pitched voices. Feeble voices are also badly conducted.

(*b.*) The size of the tube (the bronchus), and therefore the size of the vibrating column of air within it. As a consequence vocal fremitus is usually better marked on the right than on the left side, the right bronchus being the larger.

(*c.*) The distance of the part from the larynx—that is, from the point where the vibrations have their origin: the greater the distance the feebler the fremitus, and *vice versa*, so it is more marked over the upper part of the anterior aspect of the chest than the lower.

(*d.*) The thickness of the chest wall: the thicker the chest the feebler the fremitus. A thick wall, whether due to fat, muscle, or œdema, obstructs the passage of the vibrations to the surface.

From the foregoing it is apparent that too much importance must not be attached to want of success in obtaining fremitus, unless there is corroborative evidence from the other methods of investigation that it is the result of a morbid condition.

The conditions in which vocal fremitus departs from the normal may be classed as follows :—

Diminished in—

Persons with feeble or high-pitched voices (may be quite imperceptible).
Persons with thick chest walls.
Adherent and thickened pleura.
Bronchitis, } from obstruction in the bronchial tubes
Asthma, } to the passage of the vibrations.

Absent in—

Hydrothorax.
Pleurisy with effusion.
Empyema.
Pneumothorax.
Pressure of a tumour on a primary bronchus.
Anything within the bronchi blocking them, and thus preventing the passage of vibrations to the part supplied by the blocked tubes.
Phthisis, when the bronchi are plugged as in the preceding.
Adhesion with great thickening of the pleura.

Increased in—

Consolidation of the pulmonary parenchyma, as in pneumonia. It is, however, necessary that the principal bronchus going to the consolidated area be not plugged; if it is the vibrations are arrested.

Pulmonary cavities, if they have dense walls, are large and near the surface.

Solid tumours in contact with large bronchi, and also with the parietes.

Friction or pleural fremitus is the sensation communicated to the hand in cases of pleurisy where the friction is unusually coarse. It is a creaking, rubbing, or grating sensation, and is only occasionally present. It is a phenomenon of no special clinical significance.

Bronchial Fremitus.—In bronchitis and asthma, owing to local interferences with the lumen of the bronchi, the passage of air is interrupted, and sounds are produced which are known as rhonchi: they are described later. When, in these conditions, the hand is placed over the larger bronchi in front or behind, the vibrations producing the sounds are very distinctly felt, and bear the name of bronchial fremitus.

III.—PERCUSSION AND SENSE OF RESISTANCE.

Percussion.—The percussion sound of the lungs is usually defined as clear or resonant. Its precise character cannot be described; it can only be appreciated by carefully training the ear to recognise it and its various modifications in different individuals, and at different parts of the thorax. The terms, resonant and clear, are as a rule used as synonymous. It is, indeed, usual to distinguish between greater and less degrees of resonance, and to refer to the percussion sound of the thorax as increased, diminished, or impaired in resonance, or as non-resonant.

The use of the term resonant is, however, unfortunate, as it is applied in a sense which is not allowed by acoustics. It might, indeed, be desirable to discard it altogether from these pages, were it not so commonly in use that it requires at least to be recognised, and its common meaning specified.

The term resonant, then, is, as has been stated, applied to the percussion sound of the normal lung; it is a somewhat full sound, and conveys the impression of being produced by the vibrations of a structure of considerable size and containing air. The term hyper-resonant is often applied to the lung sound when it is clearer than normal, or when it approaches the tympanitic character; on the other hand, the term diminished resonance is applied when the sound is duller than normal; and the term non-resonant, or absence of resonance, when the sound is quite dull. This use of the term will be avoided in these pages as much as possible, and the student is advised to abandon its use as far as practicable.

The pulmonary percussion sound depends on the four following factors:—

1. *The thickness of the chest wall.*—The greater the thickness the duller is the percussion sound, and *vice versa.* The thickness may be due to great muscularity, to the deposition of fat, to œdema, or, as in women, to the mammæ. On the other hand, the chest wall is thin in spare persons, and in exhausting diseases.

2. *The resilience of its bony and cartilaginous framework.*—This is greater in children than in adults, and the percussion sound is clearer in the former than in the latter.

3. *The amount of air within the chest—that is, in the bronchi and pulmonary alveoli.*—When the air cells are filled with solid exudation as in pneumonia, or when the lung is compressed and the air driven out of it by the pouring out of fluid into the pleural cavity, as in pleurisy and hydrothorax, the percussion sound is dull. The percussion sound is also duller over the heart than where there is only lung underneath the part percussed, for the heart acts as a solid body.

4. *The state of tension of the lung tissue.*—If from any cause the lung tissue—that is, the walls of the alveoli—has its tension diminished, the percussion sound is modified and be-

comes tympanitic. This modification frequently occurs above the limit of a pleural effusion; in pneumonia, before the lung has become solid; in œdema of the lungs; and in caseous infiltration of the lung tissue, the air not being entirely excluded.

To understand the cause of this tympanitic sound, it is necessary to know that if a lung be percussed after its removal from the body, when it has of course partially collapsed, the sound emitted is tympanitic. It is from this, and from a consideration of the changes present in those morbid conditions in which it may be elicited, that the tympanitic sound is regarded as due to diminution in the tension of the lung tissue, and a diminution in the amount of air in the alveoli. These conditions are present in the morbid states enumerated above. The tension or tonicity is of course lessened by the inflammatory action in a pneumonia and by the pressure of the fluid in pleuritic effusion, while the amount of air in the alveoli is necessarily reduced in all cases where an exudation takes place into them. It must, however, be acknowledged that this explanation is not altogether satisfactory.

Method.

When the anterior aspect of the thorax is to be percussed the patient should be recumbent and unconstrained, or in the sitting posture.

Care must be taken that the finger is laid quite flat on the surface, and the percussion sound on one side is to be compared with that on the other in corresponding spaces and over corresponding ribs.

The apex is percussed above the clavicle by the mediate method, then the clavicles are percussed immediately, after which the rest of the lung is percussed downwards. In the first instance it is only necessary to percuss the interspaces. If an area be found where the percussion sound is altered, it can be investigated rib by rib and space by space. On no account ought the student to compare an interspace on one side with a rib on the other; and the finger ought never to rest partly on a space and partly on the adjoining ribs. The percussion sound over the ribs differs considerably from that in the interspaces; and further, if the finger is laid vertically over both, there is apt to be a space between the finger and the interspace, which materially modifies the sound, and is very likely to mislead.

It is desirable to lay emphasis on one other point, and that is, the necessity of keeping well out from the edge of the sternum: the mammary line is a good one as far down as the third rib, but from there it is necessary to diverge quite outside this line. This is necessary owing to the position of the heart on the left side, which of course affects the percussion sound, and when studying the percussion of the lungs this source of confusion is to be avoided. On the right side it is necessary to keep well out from the sternum, as the presence of the arch of the aorta, the descending vena cava, and, lower down, a distended right auricle, modify the sound. We would lay stress on this, as we have found inattention to these simple matters a very common hindrance to the student. Under percussion of the heart and the vessels at its base, it was pointed out that the outer limit of these structures was indicated by a moderate although definite alteration in the percussion sound as followed from without inwards. The alteration in the sound consists in a heightening of its pitch, caused by the thinness of the anterior edge of both lungs where they overlap the heart and large vessels. This thin edge of lung presents a smaller or shallower structure in which vibrations are set up, the result being that the pitch is raised. If the sound is further analysed, it is observed that the sound conveys the idea of being produced by the vibrations of a structure of less volume than that of the thick part of the lung, and from this the term *empty* has been applied to it by Skoda to distinguish it from that heard over the substance of the lung, and termed by the same authority *full.* The term *shallow* would be preferable to empty. In fact, the more general use of the terms shallow and full instead of reference to pitch might be desirable. This rise in pitch, or more shallow character, of the percussion sound should also be studied by percussing the right lung from above downwards. At the lower part, from about the level of the fourth rib, the percussion sound is higher in pitch, and shallower than above that limit: this is due to the diminished depth (applying depth in percussion of the thorax to the antero-posterior volume of the lung) of the lung here, and to the presence of the solid liver behind it. The same limit is known as the line of *comparative dulness* of the liver. The accompanying diagram, representing a vertical transverse section, shows the relations of parts which lead to the rise in pitch, or shallowing of the percussion sound, before the absolutely dull sound of the liver is obtained when we pass entirely from lung.

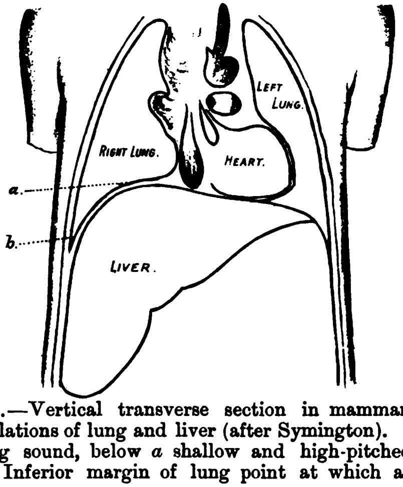


Fig. 50.—Vertical transverse section in mammary line, showing relations of lung and liver (after Symington). Above *a* full lung sound, below *a* shallow and high-pitched lung sound. *b* Inferior margin of lung point at which absolute liver dulness commences.

At the upper part anteriorly it is often necessary to percuss the whole length of the interspaces, as dulness in this position may be confined either to the inner or the outer part of them, according to the part of the apex affected. In other cases small areas of dulness may be made out scattered over the chest, but this is as a rule difficult of detection, even to a very expert percusser.

When the posterior aspect of the thorax is to be percussed, it is desirable to have the patient in the sitting posture when practicable, and when it does not subject him to too much fatigue. The patient's arms ought to be folded in front. The method to be pursued is the same as that already described for the anterior aspect. We begin above the scapulæ and proceed downwards, comparing the corresponding areas on the two sides. The sound differs much from the sound in front, owing to the difference in the structures overlying the lung: from the angle of the scapula downwards the sound, however, more nearly resembles that produced in front. To make out the extreme inferior limit of the lung, it is necessary to percuss very lightly, and there are few points of greater practical importance than the acquirement of facility in doing this, and of fully appreciating the evidence as to the exact position of the lower margin of the lung here. The reason of this is that moderate degrees of hydrothorax are exceedingly common, and it is often of the utmost importance to recognise the condition.

The percussion of the axillary and infra-axillary regions is often of great importance, and ought not to be overlooked. To investigate them the patient may sit, or lie on the side, the arm being raised with the hand over the head.

Abnormal Percussion Sounds.

These may be classed in three groups, according as the sounds are duller or clearer than normal, or are of special character.

A. *Dull sounds* are divided into—

1. *Slight dulness*—also called *comparative dulness*—when it

is slightly duller than at the corresponding spot on the other side. This is also spoken of as *impaired or deficient resonance.*

2. *Moderate dulness*—when the sound is decidedly dull, but not so marked as the next.
3. *Absolute dulness* — when the part gives a sound resembling that of a solid organ.

B. *Abnormally clear sounds* (called also *Hyper-resonant*).

These may be divided according to their degree into—

1. Slight or comparative increase of clearness.
2. Moderate and marked increase of clearness.

C. *Percussion sounds of special quality or character* :—

1. Tympanitic.
2. Amphoric or metallic.
3. Cracked-pot.

We may now consider these in order.

A. *Dull Sounds.*

1. *Slight or comparative dulness.*—This is frequently present at one or other apex, more commonly at the right. It is sometimes regarded as physiological, but this is doubtful, for it will be found that, in at least a large proportion of cases, it is associated with and due to adhesion and thickening of the pleura. A corresponding degree of dulness can be made out over other parts of the lung where the same conditions are present. To appreciate this difference percussion must not be strong; indeed, for all the finer degrees of sound, it is necessary that the student should acquire the art of percussing with one finger. Slight dulness is also present in commencing tubercular disease at the apices.

2. *Moderate dulness* is present in tubercular disease of the apices when the whole lung tissue has not become infiltrated. It is also present over patches of catarrhal pneumonia which reach the surface of the lung; and in the

patchy acute pneumonia of old people; also in pulmonary congestion in which exudation takes place into the alveoli.

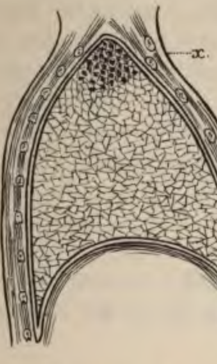


FIG. 51.—Diagram showing at *x* moderate dulness over tubercular infiltration.

3. *Absolute dulness* is present when air is completely absent from the part percussed: it is therefore present in acute

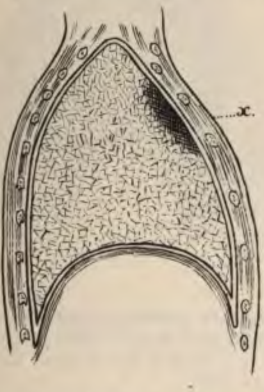


FIG. 52.—Diagram showing at *x* moderate dulness over superficial pneumonic patch.

croupous pneumonia in the stage of hepatization, when the alveoli are filled with coagulated exudation. It may also be present in phthisis from an analogous condition coupled with thickening of the pleura. It is also present over effusion into the pleura, whether the result of pleurisy or a simple hydrothorax; also over tumours, the more common ones being mediastinal tumours extending forwards and laterally, and coming into contact with the thoracic wall. Tumours of the lung itself, when of considerable size and situated at the surface, also give a dull sound.

Pitch.

The *pitch* of the sound is an element which has also to be considered at this stage, for to the student it often complicates the determination of the character of the sound. In the percussion of the lungs the pitch is, in the majority of

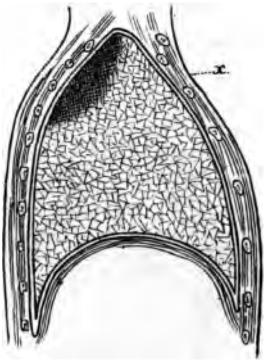


FIG. 53.—Diagram showing heightening of pitch anteriorly from consolidation posteriorly. The shaded part is the consolidated part; *x* indicates the position where the percussion sound is raised in pitch.

instances disregarded, as the dulness is the most obvious characteristic of the sound. In many cases, however, the pitch is of diagnostic value. If at any part of the chest the column of air under the spot percussed is diminished as compared with the corresponding part on the opposite side, the pitch is raised. The sound is not really a dull one in the popular sense, although the term dull is commonly applied to it; it is distinctly a *shallower* sound, that is, it conveys the impression of being produced in a part the internal capacity of which is not so large as at the corresponding part on the opposite side. We have noted this at one apex anteriorly when the posterior part of the upper lobe was the seat of pneumonic infiltration, as represented in the preceding diagram. It is also frequently present over the anterior aspect of the lower part of the chest when there is fluid in the pleura behind, as in the above diagram. In the normal condition this raising of pitch, or change from a

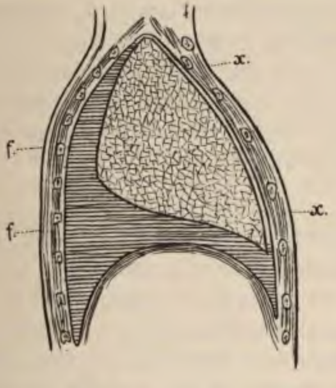


FIG. 54.—Diagram showing raising of pitch anteriorly at *x* from the presence of fluid posteriorly; at *f* the physical signs of hydrothorax.

full to a shallow sound, is noted over the anterior edge of both lungs, and inferiorly over the right one, as has been already indicated at page 132.

B. *Abnormally clear sounds*, also called *Hyper-resonant*.

1. *Slight or comparative increase in clearness* occurs from the slighter degrees of emphysema, and is not of much practical importance.

2. *Moderate and marked increase* may be classed together, as they necessarily merge into each other, the dividing line being purely empirical, and depending on the individual observer. It is present all over the chest when both lungs are emphysematous throughout. When the emphysema is confined to the edges of the lungs the alteration in the percussion sound is confined to those parts. This can very frequently be noted over the front of the chest, the percussion sound becoming distinctly clearer on percussing from without inwards,—that is, towards the edge of the lungs. It is also present over the area of superficial cardiac dulness, when the heart is covered by the emphysematous border of the left lung. The more markedly clear sounds merge into the tympanitic variety.

C. *Percussion Sounds of special Quality or Character.*

1. *Tympanitic.*—This is the sound which is elicited by percussing the stomach or intestines when they contain air. In the normal condition it is not produced by percussion of the thorax, save inferiorly on the left side, when the stomach is dilated, as is specially referred to under the examination of that organ. In several morbid conditions, however, the percussion sound presents this character. They are as follows:—

(*a.*) Pneumothorax, *i.e.*, the presence of air in the pleural cavity, leading to retraction of the lung. If the tension of the air within the pleura be great, the percussion sound retains its tympanitic character but rises in pitch, and is commonly referred to as duller. This is also true of the percussion of the stomach and intestines: if their walls be tightly stretched by the amount of contained gas, the tympanitic sound they give rises in pitch.

(*b.*) Large and superficial cavities in the lung substance, the result of destructive changes.

(*c.*) The early stage of pneumonia before consolidation takes place. This is generally regarded as due to diminished tension in the affected portion of lung, as explained at p. 130.

(*d.*) Œdema of the lungs, the physical conditions being much the same as in *c.*

(*e.*) Over the lung, immediately above pleuritic effusion. The explanation of this is similar to that given under *c*, namely, that the tension of the lung is diminished above the limit of the effusion. The fact of a tympanitic sound being present in this condition is to be remembered, for it is sometimes regarded as proof of the presence of pneumothorax.

(*f.*) "The tracheal resonance of Williams" is the term which has been applied to a tympanitic sound present at the apex of the left lung, close to the sternum, when the lung there is consolidated. To understand this it is necessary to note that the percussion sound over the larynx and trachea is tympanitic, the pitch depending on whether the mouth is open or shut. This sound is lost over the bifurcation of the trachea and over the primary bronchi, owing to the interposition of pulmonary tissue; when, however, the intervening lung is consolidated, either from exudation into the vesicles, or from compression, the sound may be elicited owing to the better conduction of the vibrations by the solid tissue.

2. *Amphoric or metallic* percussion sound. This sound is tympanitic in character, but has in addition a metallic clang or echo. In other words, the sound is prolonged, the prolongation being compared to an echo. It is comparable to the sonorous prolongation of the voice, which is observable when we speak in an empty hall; it is also to be noted when

a large, empty or partially filled, and covered vessel is percussed. The tympanitic sound, on the other hand, is not prolonged, it ceases immediately after the percussion blow producing it. The amphoric sound is heard best in cases of pneumothorax and in large superficial cavities in the lung ; in both it is necessary that the walls of the cavity be smooth, so that the sound waves may be reflected equally from all parts of the wall. In pulmonary cavities the sound comes out best when the mouth is open.

3. *Cracked-pot Percussion Sound* (*Bruit de pot fêlé*).—This sound, as its name implies, resembles that produced by percussing a cracked vessel. It can be simulated by clasping the hands at right angles to each other, and then striking them smartly against the knee. It is produced—

(*a.*) Over pulmonary cavities when they communicate directly with a bronchus, and when the chest wall overlying them is emaciated and yields to the percussion stroke. It is almost confined to cavities at the apex. To elicit the sound percussion should be performed during expiration, and the patient's mouth ought to be open. It is caused by the sudden expulsion of a portion of air from the cavity into the bronchus. While this is the most common condition in which the sound is obtained, there are several others in which it is occasionally present, namely—

(*b.*) In some cases of pleurisy above the limit of the effusion, due probably to the yielding of the chest wall, and the partially compressed state of the lung favouring the sudden expulsion of air by the percussion stroke.

(*c.*) In pneumonia before consolidation has taken place. In both this and the preceding condition, it will be remembered that a tympanitic percussion sound is sometimes present.

(*d.*) When an opening exists through the parietes into the pleural cavity. It can therefore be readily demonstrated when the pleura has been incised for empyema, and its non-production when the opening is artificially closed can also be demonstrated.

(*e.*) In pneumothorax, where there is a free communication between the cavity and a bronchus.

(*f.*) It is readily produced in children over the upper part of the lung, especially when crying, owing to the very yielding character of the thoracic wall in infancy.

In all these the physical explanation is the same, and it is only necessary to specially warn students against assuming that a cracked-pot sound necessarily implies the presence of a cavity; and this warning is all the more necessary as there is probably no evidence derived from percussion, unless it be the tympanitic sound, which is more frequently misconstrued.

The Sense of Resistance.

Before leaving the consideration of percussion, it is necessary to refer to the sense of resistance. It may be explained as follows.

When a solid body or a distended viscus is percussed mediately, there is a feeling of resistance which indicates that the body is solid. On the other hand, structures containing air do not give the same sense of resistance unless the walls overlying them be very thick. This feeling or sense of resistance is of course quite apart from sound, and is a guide without the sense of hearing. In percussion the power of recognising variations in the sense of resistance becomes almost unconsciously very highly developed, and enters to a much greater extent than is generally recognised into the determination of whether a part is dull, and which of two points is the duller. So much have we been conscious of this, that we have often found it necessary to use a pleximeter, other than the finger, when we wanted simply to analyse the characters of the sound, and to be wholly unbiassed by this additional element. From a purely practical standpoint, it comes therefore to be of decided value in percussion. From what has been said above, it will be understood that it varies with the degree of solidity of the part percussed, and therefore is in direct ratio to the degree of dulness; that is, the greater the dulness the greater the sense of resistance.

IV.—AUSCULTATION.

One of the greatest difficulties in the auscultation of the lungs is the variety of classification of the sounds, and the want of agreement amongst teaching physicians as to the characters which distinguish the different groups. This necessarily leads to great confusion in the mind of the student, and surrounds the exposition of the subject with difficulties, while it is, we fear, somewhat hopeless to attempt to meet the views of all teachers of practical medicine. There is not by any means the same degree of confusion in the interpretation of the signs, but even here there is room for greater accuracy, and the attainment of this can only be realised by the more rigid definition of the terms at present in use. In teaching and in learning, the tendency, we think, is too much towards classification without descriptive and critical examination of the sounds, which would constitute a training in itself. The practice is too much to draw attention to the breathing at a specified spot in an individual case, as bronchial, tubular, broncho-vesicular, or whatever else the individual teacher may choose to call it, without that critical analysis of the sound which would resolve it into the component elements which give it its special features, and on some of which at least its classification must be consciously or unconsciously based. That this must necessarily be the case is easily rendered apparent. Respiration consists of two parts, inspiration and expiration. Over different parts of the respiratory tract the audibility of inspiration and expiration varies both in length, loudness, and tone, and the relative length of the one to the other. A knowledge of these variations, as they are heard over different parts in the normal condition, is necessary to the recognition of the alterations which occur in disease, but even in disease all alterations must be confined to inspiration and expiration, and the only modifications they can manifest must fall under loudness, character, pitch, and the relative length of inspiration and expiration to each other. In disease there are in addition accessory sounds which have to be considered.

Auscultation in the normal state.

Normal respiration consists, as has been already said, of inspiration and expiration. During each a sound is produced, and it is on this fact that auscultation of the respiratory organs is practicable.

If auscultation be practised over the trachea, or, what is still better, over the cervical vertebræ, where the pressure of the stethoscope is less disagreeable to the person auscultated, it will be found that a sound is heard during inspiration and another during expiration. The loudness and tone or character of these sounds depend on whether the individual auscultated is breathing through the nose only, or has the mouth open and is breathing mainly through it, and on whether the breathing is slow or rapid.

If the breathing be performed with the mouth open and is not accelerated, it will be observed (*a*) that the inspiratory and expiratory sounds are distinct from each other, and separated by a short but distinct break; (*b*) that they are about equal in duration; (*c*) that they have a soft blowing character, which is more pronounced and louder during the expiratory than the inspiratory part.

If now the mouth be closed and respiration carried on through its proper channel, the nose, it will be observed that as regards (*a*) and (*b*) the sounds are the same as when the mouth was open, but that an important change has occurred as regards (*c*). It consists in an alteration in the loudness and in the character of the sounds,—the loudness has become intensified and the sound has become harsher.

In the first case the sound heard was produced by the air passing into and out of the mouth, and by its passing in and out past the vocal cords, where the lumen of the air passage is smaller than above and below them. In the second case the greater loudness and harshness were due to the air passing in and out by the nose. Owing to its construction, a narrow passage is formed opening into a wider one, the pharynx, and this gives the conditions which intensify sound; there are other elements in this, which need not, however, be dwelt upon.

We have also stated that the character of the sounds depends on the rapidity of the breathing. If the breathing be rapid and conducted by the mouth, the sounds are louder and harsher than when breathing is slow. This is due to the greater friction resulting from increased speed, the friction occurring at the vocal cords mainly. In this condition the sounds approximate to those heard when the breathing was nasal.

In these three cases the sounds are heard over the regions specified as they are conducted there by the air in the trachea, or transmitted to the vertebræ from the trachea. They are audible anteriorly until the sternum is reached, and posteriorly as far as the upper dorsal vertebræ. The sound would of course be heard over the large bronchi were they within reach of the stethoscope, but they are so overlaid by the parenchyma of the lungs that sounds conveyed into them from the larynx and trachea have to pass through it before reaching the surface, and in doing so they are considerably modified. As will be seen presently, there is a type of breathing which is known as "bronchial," as it resembles what was supposed to be heard over the bronchi. We have, however, seen that the bronchi, even in their primary divisions, are so covered by lung that the sound in them can only be heard after it has been modified by its passage through lung parenchyma. We take therefore the sound heard over the trachea and spine as the type of what is clinically known as *bronchial breathing.*

If now auscultation be practised over the thorax at any part where the lung comes into contact with the parietes, it will be observed that the sounds have greatly altered. The first thing that as a rule impresses the student is the difficulty of hearing anything, the sounds being so much feebler than over the trachea. Instead of the more or less harsh or blowing sound during inspiration and expiration, there is now an extremely soft and often a very feeble sound heard during inspiration, which runs into a still feebler sound synchronous with the first third or so of expiration. The relative lengths of the movements of inspiration and expiration may be expressed thus—

Inspiration : Expiration : : 5 : 6,

Whereas the relative duration of the sounds is—

Inspiration : Expiration : : 3 : 1.

But while this is the typical relation, the expiratory part is in many individuals quite inaudible. *Vesicular breathing* is the term applied to breath sounds of this type; it has been compared to the rustling of leaves, and is often referred to as breezy in character. There is still a difference of opinion as to whether this sound is produced by the air passing into and out of the air vesicles, or is due simply to the conduction downwards of the tracheal sound, the tissues through which it has to pass modifying its character. We believe the latter to be, in the main, the correct view, and abnormal auscultatory signs are more easily explained by it.

While these are the sounds heard at the upper and lower parts of the respiratory system, there is present in some individuals at certain points what must be regarded as a transition sound. When present it is best heard in the interscapular region, about the level of the spines of the scapulæ. Its intermediate character is shown (*a*) by the soft blowing character of the inspiration, or by an inspiration rather harsher than that heard over the body of the lungs; (*b*) by an expiration which is neither as long nor as harsh as that heard higher up; while (*c*) the relative lengths of inspiration and expiration approximate more nearly to those present over the parenchyma than over the trachea. From its intermediate character it has been called *broncho-vesicular* or *indeterminate* breathing, the former being the better term. Its explanation is that the large bronchi are nearer the chest wall here than at any other part, but that even here the sounds conducted into them from above have to pass through a considerable thickness of lung tissue to reach the chest wall, and therefore the ear; in their passage they are *damped down.*

The term broncho-vesicular would be equally applicable did we hold the opinion that the air vesicles themselves produced sound during their inflation and retraction, for the sound would then be a combination of the bronchial sound conducted from a distance, and the vesicular sound produced at the spot.

The foregoing is simply a statement of fact, the phenomena having the names in general use applied to them. The cause of the unsatisfactory state, not of the nomenclature so much as of the individual application of it, necessitates some explanation, and thereby what appears almost hopeless confusion to the student may be simplified and put somewhat in order.

In the first place, it is found that the term "bronchial" is applied by some to all the variety of sound, described in the preceding pages as audible over the trachea, cervical vertebræ, and interscapular regions. This includes breathing characterised at one end by harsh inspiration and expiration of equal length, to, at the other end, breathing with a soft blowing inspiration, followed by a short expiration which varies in tone. Not only that, but breathing the inspiration of which shows any of these characters, and is followed by an inaudible expiration, is also classed by some as bronchial. To meet the difficulty raised by including such a variety of sound under one term, some use the term *tubular* to denote the type of harsh sound heard over the trachea; but this only meets a small part of the difficulty.

On the other hand, some exclude from the term "bronchial" all sounds which have not both the harsh inspiration and the long harsh expiration heard over the trachea with the mouth shut; or extend it also to a breathing in which the expiration is as long as the inspiration, but is blowing in character instead of harsh.

The confusion which must necessarily result is apparent, and there is little probability of advance in accuracy in the auscultation of the lungs until something is done to clear it up. As a matter of fact we have hardly made any advance on Laennec's work, and this is scarcely to be wondered at, when it is realised that the auscultatory phenomena recorded by one observer convey no sufficiently definite and distinctive impression to the mind of another to enable him to reproduce in imagination the sounds which were heard.

It is then of the first importance that the student should analyse the sounds to which he is listening. He should, *first*, separate inspiration from expiration; *second*, note their relative lengths; *third*, note the character of inspiration and of expiration, whether soft, or harsh, or blowing, or a combination of these. By so doing he will be able readily enough to adopt any classification necessary for the present, and it will tend to make him an independent observer in the future.

Having thus considered the phenomena to be noted in auscultation of the respiratory organs in a state of health, we have next to turn our attention to the phenomena in diseased conditions.

Auscultation in Abnormal States.

Auscultation in abnormal states is practically confined to those parts of the chest which cover the lungs, where in fact vesicular breathing, or, as in the interscapular regions, broncho-vesicular breathing is normally present.

A. *Varieties of Vesicular Breathing.*

Vesicular breathing is liable to various modifications, which, however, are not sufficient to remove them from the rank of vesicular: they are classed as follows:—

Weak vesicular.
Harsh do. or puerile.
Do. do. with prolonged expiration.
Wavy, jerking, or interrupted vesicular.
Absence of breathing.

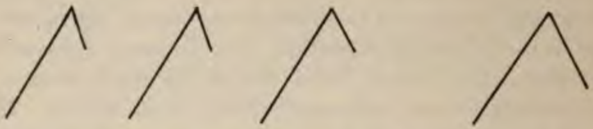
(*a.*) *Weak or faint vesicular.*—In this variety the relation of expiration to inspiration is maintained, but the sounds are faint. It is present in persons whose lungs are voluminous, and who, from the extent of breathing surface they possess, breathe correspondingly slowly and quietly. It is also, however, present over a part of the lung where there are pleural adhesions, or a thin layer of fluid between the lung and the chest wall.

(*b.*) *Harsh vesicular or puerile.*—The normal relation of expiration to inspiration is maintained here also, but each is louder and harsher than over the normal adult lung. It is called puerile, as it is the character which the vesicular breathing normally presents in children. It is present in the adult when the function of a considerable area of lung is in abeyance. It is therefore present over the unaffected side when there is large pleural effusion on the other side. If there be moderate effusion into both pleuræ, as is often the case in cardiac disease with failure of compensation, it is present over the upper part of both lungs. It is also present over the unaffected lung in pneumonia, over the upper parts of both when there is congestion at the bases, and in fact over the unaffected part in all conditions where, as has been said, the respiratory function of part is in abeyance, or has been permanently destroyed or seriously lowered. It is therefore also present over unaffected parts in phthisis.

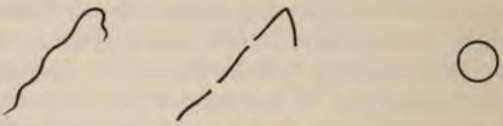
(*c.*) *Harsh vesicular, with prolonged expiration.*—The expiration in this variety is, as its name implies, longer than in the preceding or puerile variety. The character of inspiration and expiration is the same as in puerile. It is present in many individuals at one or other apex, commonly the right, and is regarded by some as a normal condition. This, however, is exceedingly doubtful, and so far we incline to hold that in many of these cases—we cannot say in all—there is some morbid alteration, not, however, necessarily indicating active change, but a change the result of an old inflammatory process, and usually fibroid in its nature. The great frequency with which such changes are seen in the *post-mortem* room tends to support this view.

(*d.*) *Wavy, jerking, or interrupted vesicular, or cog-wheel respiration.*—Here the inspiration, instead of being continuous and sustained, is, as its name implies, wavy, jerking, or interrupted. Its character is best understood by reference to the subjoined diagram. It is present in nervous patients, especially when they make efforts to breathe deeply at the request of the auscultator. It is, however, also present in early tubercular infiltration at the apex. In the first it is due to the irregular and undecided manner in which the respiration is performed, while in the second it is probably due to breaks or delays in the passage of the air, and therefore of the sound, passing affected parts.

(*e.*) *Absence of vesicular breathing.*—This is noted in conditions in which conduction of the respiratory sounds is no longer possible. In its most marked and typical form it is noted in effusions into the pleura with compression of the lung, whether the fluid be serous, purulent, or hæmorrhagic. It is also present in pneumothorax, with collapse of the lung, if the opening between the lung and the pleural cavity be closed. The absence of breathing over an apex is sometimes to be noted in cases of phthisis, even though cavity be present. This is exceedingly misleading to the student, and sometimes to more experienced auscultators. It is due to the bronchi of the affected part being filled with secretion, whereby the conduction of the sound is interrupted; over cavities it may be in part due to this, and in part to the cavities themselves being filled with viscid or more fluid contents. Coughing, however, usually elicits sounds which indicate the condition of the part.



Normal. Weak or faint. Harsh or puerile. Harsh with prolonged expiration.



Wavy. Jerking or interrupted. (Cog-wheel.) Absence.

FIG. 55.—Diagrammatic representation of varieties of vesicular breathing. (After Wyllie.)

B. *Bronchial Breathing.*

In morbid conditions, which will be referred to presently, bronchial breathing is present over those parts of the chest where normally the character of the breathing is vesicular.

The characters which separate it from vesicular have been already considered, and the indefiniteness of the term as in general use has been referred to; it is, however, desirable to describe more fully the various varieties of breathing sounds heard over the pulmonary parenchyma which have been included under this head.

First. The term is applied to breath sounds characterised by an inspiration and expiration equal in length, separated, however, by a break, and each as harsh, loud, and high pitched as the harsh sound heard over the trachea during nasal breathing. This sound is imitated by putting the tongue in a position for pronouncing the "guttural" *ch* and then breathing deeply. Not only, however, may the sound be as loud and harsh as this, but it may even be more intense : this, for instance, is common in croupous pneumonia when the affected part is consolidated. This variety is so typically present in pneumonic consolidation that it is termed *tubular breathing* by many authorities. Were the term strictly confined to this special variety of bronchial breathing no objections might be made to it, but practically it is found that its boundaries are modified by individual observers, so that even it may not convey a sufficiently definite conception to another. Further, there is some confusion as to the physical explanation of the sound which the term perhaps tends to countenance, for the idea in many minds is that the sound has its origin in the consolidated part. The correct explanation is that the sounds heard over the trachea are propagated into the bronchi, and are not damped down (to repeat a term used in this relation already) by having to pass through the normal spongy lung tissue before reaching the chest wall, but are conducted in all their intensity through the solidified tissue. Not only is the sound not diminished, but it is often very markedly intensified, as can be verified by auscultating over the trachea, and comparing the sound there with that over the consolidated part. The reason of this intensification is that the consolidated lung acts as a resonator.

If the bronchi are plugged with consolidation the conduction of the sounds is impeded.

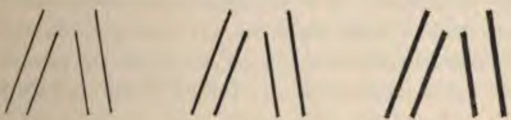


FIG. 56.—Bronchial Breathing—various degrees of loudness. (Modified from Wyllie.)

This variety may be represented diagrammatically as above, its loudness being represented by the thickness of the lines, for the intensity varies within somewhat wide limits.

Second. The term bronchial is applied to a variety of breath sound characterised by its distinctly blowing character, and which has not the harsh character of the preceding variety. In this form expiration may be as long as inspiration, but is often shorter; while in some cases the expiration is longer, louder, and more blowing than inspiration. There is a break between inspiration and expiration. This and the preceding variety merge into each other, and combine in various ways. They may be represented thus—

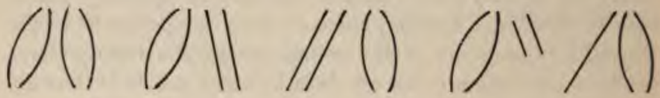


FIG. 57.—Bronchial breathing of blowing character.

Third. There is a variety of breathing which is known as *cavernous.* It is heard in many persons with great distinctness over the box of the larynx, and in some over the trachea. It, however, varies in distinctness, and is not in all equally characteristic. It is a low-pitched blowing sound of considerable volume; inspiration and expiration are about equal in length, and there is a break between them. This sound is more voluminous and hollow in character than the sound usually heard over the trachea or cervical vertebræ. From its hollow tone it suggests to the mind of the observer the name it bears—cavernous. It is the same sound, produced mainly in the mouth and nose, which we have already described, and it has its special tone over the larynx from its proximity to the main seat of its production. It may be represented thus—

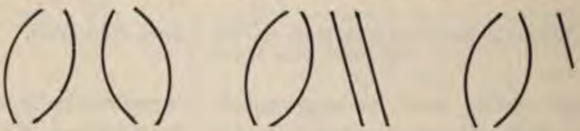


FIG. 58.—Diagrammatic representation of cavernous breathing.

This form has been placed under the head of bronchial, for it is the bronchial sound heard nearer its seat of production, and also because it becomes purely a matter of individual habit or individual fancy whether sounds which are on the borderland of this and the preceding are described as bronchial or cavernous, some observers classing some of the modifications of the second variety as cavernous.

From the foregoing it is seen that the varieties of bronchial breathing depend on the *character of the sound*, while in all save one the *length of the expiration* is diagrammatically represented as equal to inspiration. While these two factors are present in typical instances, there are cases in which expiration is not so much prolonged, and in which, therefore, the tracheal sound, which is the standard, is not reproduced in all its characters. On this point hangs another element of confusion, some refusing to call the breathing bronchial because the expiration is not prolonged, while others unhesitatingly class it as bronchial because the *character* of the inspiration corresponds with the character of the tracheal sound.

Bronchial breathing either in its harsh or blowing variety is heard over consolidated lung, the lung in this condition acting as a good conductor of the tracheal sound, or, as has been already said, even as a resonator,—that is, an intensifier of the sound. It is therefore present over the affected area in croupous pneumonia and in tubercular infiltration and consolidation. It is also present in fibroid induration of the lung. In pleuritic effusion it is present over the compressed part of the lung above the limit of the effusion, and is readily mistaken for pneumonia when the level of the fluid is low. It is also present over an area of collapse.

Bronchial breathing, or its cavernous variety, is also present over phthisical cavities or bronchiectatic dilatations.

C. *Broncho-Vesicular, Vesiculo-Bronchial, or Indeterminate Breathing.*

Some of the breath sounds referred to in the last paragraph, and which some clinicians class as bronchial, ought properly to be classed under this head, for they present neither the characters of the standard bronchial nor vesicular class. On the other hand, the sounds sometimes included in this class inevitably lead to misunderstanding and confusion.

The above terms ought to be confined to sounds which are midway between bronchial and vesicular; or to sounds in which the inspiratory or the expiratory part has more or less of the character of some of the varieties of bronchial, but in which the other half retains the vesicular character. In cases, however, in which the inspiration may be cavernous or harsh bronchial, while the expiration may have nothing to distinguish it from vesicular, the breathing might be described as presenting cavernous or harsh bronchial inspiration, with expiration harsh or soft and not prolonged, or inaudible, as the case may be, and thus classification would be avoided. They are frequently met with, and classing them misleads, or at best conveys no definite impression to the mind of any one persuing the record of the case. The great desideratum is to have a record in which there would be no difficulty in reproducing by imitation the sounds which had been heard, and at present this is impossible.

This class of breathing is heard in some persons in the interscapular region, about the level of the third dorsal vertebra, owing to the proximity of the large bronchi, and the tracheal sound not being so much modified as where the interposing pulmonary tissue is greater. It is also sometimes heard over the manubrium sterni.

In abnormal conditions it indicates partial consolidation, and may be present at the apex in the early stage of tubercular infiltration.

D. *Amphoric Breathing.*

This sound is not heard in the normal condition over any part of the respiratory system. It can be imitated by blowing into the mouth of any narrow-mouthed vessel, and is caused by the mode of reflection of the vibrations from the walls of the vessel. It varies in pitch with the dimensions of the vessel.

It is heard over large pulmonary cavities into which a bronchus opens, and in pneumothorax if the opening between the lung and the pleural cavity be patent.

It is often confused with cavernous breathing, and care should be taken to strictly confine it to those cases where its specific character is distinctly marked.

ACCOMPANIMENTS.

In addition to the modifications or alterations in the character of the breathing which have been considered in the preceding pages, there are accessory sounds present in various morbid conditions which are known as accompaniments. They are produced in four different positions—(1.) the bronchi, (2.) the alveoli, (3.) in pulmonary cavities, and (4.) in the pleura.

With the exception of the last, the term *râle* is applied to them all. Etymologically the word signifies a noise, but it is applied in a general sense to all accessory sounds in the air passages.

The various accompaniments have distinctive names as follows :—

A. *Rhonchi.*

Rhonchi are sounds of a whistling, sonorous, cooing, or squeaking character, produced in the bronchial tubes. The low-pitched ones are produced in the larger tubes, and are known as *sonorous rhonchi*, while the high-pitched ones produced in the smaller tubes are *sibilant rhonchi*. In both, the sounds are caused by (*a.*) interference with the lumen of the tubes by an unequal thickening of the mucous membrane; or (*b.*) what is more common, by the presence of mucous lying in the tubes at different points; and (*c.*) by spasmodic contraction of the bronchi. In all these the air, passing the constricted point into a wider part beyond, sets up vibrations which are heard as rhonchi, and may even be felt by the hand applied to the chest wall. They are represented in Fig. 59.

They are present either during inspiration or expiration, or both. From their character they are sometimes called ***dry râles*** to distinguish them from the next class.

Rhonchi are present in bronchitis, whether it be a simple bronchitis, or part of the pulmonic changes in phthisis, or due to congestion of the bronchial vessels from mitral lesion. They are also present in asthma, and in bronchitic asthma.

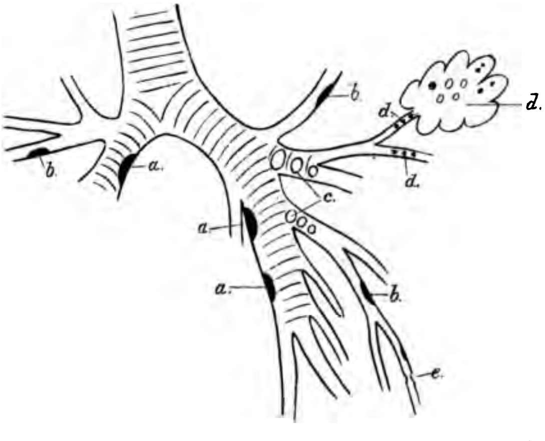


Fig. 59.—Representing the production of rhonchi, and of crepitations in the bronchi and alveoli,—at *a* rhonchi from mucus in the large bronchi; at *b* in the small tubes; at *c* coarse crepitations in large tubes; at *d* fine crepitations in small tubes and alveoli; at *e* rhonchi from spasm of small tubes.

B. *Crepitations.*

This term is applied to sounds which resemble those produced when salt is thrown into the fire, the bursting of the bubbles in boiling water, or by rubbing the hair between the fingers close to the ear. They are produced in the bronchi, in the alveoli, and in pulmonary cavities. They denote the presence of air and fluid, or viscid secretion in these positions, and are caused by (*a.*) the bursting of air bubbles as the air is drawn into or expelled from the lung, and (*b.*) by the separation of the bronchial and vesicular walls from their contents during inspiration. That the latter is capable of producing a sound resembling crepitations may be shown by pressing the points of two fingers together and separating them close to the ear.

They are sometimes called *moist râles* to distinguish them from rhonchi or dry râles. Dry and moist simply indicate the impression made on the observer's mind as to whether the sound is produced by dry or moist conditions.

Crepitations vary also in size, number, and tone or character. According to their size they are *fine*, *medium* or *subcrepitant*, and *coarse*. In character the coarser kinds may be bubbling, gurgling, metallic or tinkling. They may be represented thus—

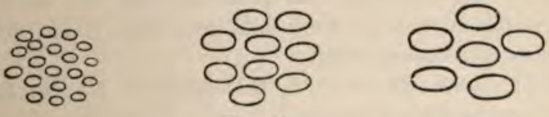


FIG. 60.

Fine Crepitations. Medium Crepitations. Coarse Crepitations.
(After Wyllie.)

They are present mainly during inspiration, but sometimes during expiration also, and the part of respiration during which they are present ought to be noted.

Fine crepitations are heard in the first and last stage of croupous pneumonia, they are usually present in very great numbers, and give the impression of uniformity in size. They are also present in œdema and congestion of the lungs, in which, however, they may be larger and belong to the subcrepitant variety. The medium or fine variety is present also in bronchitis affecting the smaller tubes. Coarse crepitations of bubbling or gurgling character are formed either in the large bronchi or in pulmonary or bronchiectatic cavities. Metallic or tinkling crepitations are so called from their metallic tone; they are produced in large pulmonary cavities, or in hydro-pneumothorax, their special character being due to the reverberation of the sound in the cavity.

C. *Friction.*

Friction is a rubbing sound caused by the movement of one inflamed surface on another. Heard over the lungs it is due to pleurisy. Its coarseness and intensity vary widely in different cases; sometimes it is so fine as to be hardly, if at all, distinguishable from fine crepitations, at other times it is a coarse, loud sound, like the creaking of thick leather. If there is doubt as to whether a sound is pleural or intra-pulmonary in origin, it can sometimes be settled by making the patient cough, when, if the sound be intra-pulmonary, it is modified, while if it be pleural, no appreciable modification results. In addition, firm pressure with the stethoscope intensifies friction in some instances, while it has no effect on intra-pulmonary sounds.

Pleuro-pericardial friction is referred to at page 59, and need not be again considered.

Other points will be found referred to at page 163 under pleurisy.

D. *Additional Sounds.*

In addition to the foregoing, there are three sounds which will only be mentioned here, as they are dealt with fully under hydro-pneumothorax at page 171. They are metallic tinkling, the bell sound, and the succussion sound.

VOCAL RESONANCE.

The signification of this term has been referred to already, and its intensity depends on the general laws enumerated at page 126.

It is liable to variations, which require to be further considered, and are the result of morbid changes in the lung and pleura, these acting either in the direction of intensifying or diminishing its loudness, or modifying its character.

Increase of vocal resonance may be so slight that it can only be described as *slight increase*; when, however, it is more decided, it is known as *bronchophony*, from its resemblance to the vocal resonance over the bronchi, as heard in the interscapular areas. It indicates that the vibrations set up in the larynx by speaking are conducted through the lung better than in the normal condition, that in fact changes have occurred in the lung which make it a better conductor of sound vibrations. Consolidation of the lung is therefore the condition in which it is most typically present; it is also present over collapsed lung, over cavities, and over a part where there are both consolidation and cavities. When very marked the resonance may be even more intense than over the trachea, this being due to the consolidated lung or the cavity acting as an intensifier of the voice sound.

Pectoriloquy is the term used to denote that intensity of vocal resonance which conveys the impression to the auscultator's ear of the voice being spoken into the mouth of the stethoscope, and which closely resembles the voice as heard on auscultating the larynx or trachea. This peculiarity is most striking when the patient whispers, and is then known as *whispering pectoriloquy*. It is present over pulmonary cavities and consolidated lung when connected with large bronchi.

Diminution or absence of vocal resonance.—Attention has already been drawn to the fact that vocal resonance is very imperfectly, if at all, distinguishable in some persons. The pathological change which, however, most commonly completely annuls it, is effusion into the pleura. It is also absent if the bronchus leading to a part be occluded, and secretion in the bronchi diminishes its intensity. In all these the vibrations are impeded and do not reach the chest wall.

Modifications in the character of the vocal resonance.—The first of these is *œgophony*, so termed from its bleating character. It is heard in some cases of pleuritic effusion, towards the upper limit of the fluid where it forms but a thin layer between the lung and the thoracic wall. Its mode of production is difficult of explanation, but it is most probably the result of an arrest or non-transmission by the fluid of the fundamental tone of the voice, while the harmonics are not arrested.

The second is *amphoric resonance*, which is fully referred to under pneumothorax at page 167. It may also be present over intra-pulmonary cavities.

THE PHYSICAL SIGNS OF SOME OF THE PRINCIPAL LUNG AFFECTIONS.

Bronchitis.

This is one of the commonest affections of the respiratory organs, and only presents distinctive features on auscultation. Inspection and percussion are not modified. On palpation bronchial fremitus can often be felt. The characteristic auscultatory signs are rhonchi, which vary in time and loudness in individual cases, and also at different times in the same case. They are, as a rule, best heard posteriorly in the inter-scapular regions and at the base. They are heard over both lungs equally. When the signs of bronchitis are limited to a part, as to one apex, it suggests a suspicion of phthisis.

Emphysema.

At page 118 the barrel shape of the chest in this condition has been referred to.

Expansion is much diminished, and in extreme cases is absent, especially at the upper part, the principal movement being an upward one of the chest as a whole, performed mainly by the action of the sternocleido-mastoid and scaleni muscles. When the patient coughs there may be marked distention of the supra-clavicular regions and of some of the intercostal spaces close to the sternum, owing to the inflation of the emphysematous lung tissue from the increased pressure during coughing.

The percussion sound is increased in clearness, and may even be somewhat tympanitic. The area of superficial dulness of the heart is diminished, and may be entirely absent, owing to the emphysematous lung interposing between the organ and the chest wall. The line of superficial dulness of the liver on the right side is lower than normal; in fact, the clear percussion sound of the lung may be present as low as the edge of the ribs. Posteriorly the clear pulmonary sound is also lower than normal. Owing to the increased bulk of the lungs, not only is the thorax distended, but the diaphragm is depressed, and consequently the liver is pushed down. The heart is also lowered in position, its axis is more vertical, and its pulsations may be visible in the epigastrium.

The signs present on auscultation have been variously described, and the want of unanimity appears to be due to the fact that the majority of cases of emphysema have at the same time bronchitis. When bronchitis is not present the breathing is vesicular in character and fainter than normal, but on the other hand, if bronchitis be present, the respiratory sounds are harsh, and there are snoring or sibilant rhonchi especially marked during expiration.

Owing to the obstruction which results in the pulmonary circulation the pulmonary second sound is accentuated, the right heart is dilated, the veins of the neck are distended, and there is marked lividity.

Pneumonia.

In the first stage (congestion and exudation) there is comparative percussion dulness, the pitch being higher than on the unaffected side; vocal fremitus is not increased; on auscultation there are vast numbers of very fine crepitations heard during inspiration, and vocal resonance is not increased. A deep breath or a cough makes the crepitations more numerous and more audible.

In the second stage (consolidation) there is absolute dulness on percussion; the vocal fremitus is increased; on auscultation the breathing is bronchial, in the most typical cases having the harsh *ch* sound, and called by some tubular, but in other cases it is more blowing: the sound is often louder than the respiratory sound heard over the trachea or

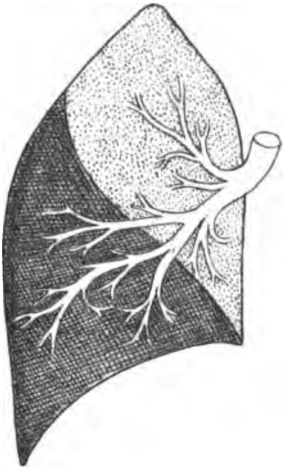


FIG. 61.—Representing pneumonia of the inferior lobe with the physical signs characteristic of consolidation.

cervical spine, the consolidated lung acting as a resonator; vocal resonance is much intensified, being bronchophonic.

In the third stage (resolution) the physical signs more resemble those of the first stage, crepitations reappear, and are often present during both inspiration and expiration. In this stage they are known as *redux crepitations.*

As a rule pneumonia affects the whole lobe of one lung, and is strictly confined to it. It is thus necessary to bear in mind the relations of the different lobes to the surface in determining the extent of lung affected. It is also, however, necessary to remember that, while this is the rule, there are many exceptions to it, and that cases are met with in which only the anterior or posterior part of a lobe is affected, while in other cases there may be a comparatively small patch, this last being especially the case in old people.

Pleurisy.

The most typical sign of pleurisy is friction. In the early stage, however, it is not always possible to hear it. This is, in part at least, due to the pain caused by inspiration, which leads the patient to use the affected part as little as possible: this, for example, is effected, when the pleurisy is at the base, by preventing the action of the diaphragm and using the upper part of the lung. It may, however, even in this case be elicited by encouraging the patient to breathe naturally or deeply, or by coughing. In not a few cases the pleurisy begins on the diaphragmatic surface of the lung when it is of course beyond the reach of auscultation.

Later, when effusion has occurred, there is dulness on percussion, absence of vocal fremitus, absence of breath sounds and of vocal resonance. By means of vocal fremitus it is as a rule possible to define very accurately the upper limit of the fluid. It is more accurate than percussion, because the compressed and airless lung above the fluid also gives a dull sound.

The physical signs which are frequently present at the uppermost part of the effusion, or immediately above it, require to be clearly kept in mind, as they frequently lead to errors in diagnosis. The œgophony which may be present has been already referred to at page 159, and is not a misleading sign. Those which mislead are—(*a*) the presence of a *tympanitic percussion sound* above the dull sound caused by the fluid. The sound is produced by the compressed but not perfectly airless lung. Its position is often determined by the pre-existence of pleural adhesions. If there are no adhesions the lung is compressed upwards and towards its root, and the tympanitic sound is immediately above the limit of the fluid; but if, for instance, the lung be adherent anteriorly, it cannot be compressed upwards, but is compressed forwards, and over the upper part of this compressed area, which is not quite airless, and is lying against the anterior aspect of the chest wall, the tympanitic sound may be present and may be mistaken for pneumothorax. (*b*) The presence of *bronchial breathing and crepitations above the fluid.* These are produced in the compressed and congested lung, and when the fluid is not very abundant the condition is often mistaken for pneumonia. This not only occurs in pleurisy, but in hydrothorax, and the signs disappear if the fluid be removed.

It is of great importance to learn to recognise the presence of moderate effusion into the pleural cavity, whether it be simply a hydrothorax or the result of pleurisy, and to do this it is necessary to be familiar with the percussion sound over the extreme bases of the lungs posteriorly, for dulness of a few fingers'-breadth in this position may indicate a very considerable effusion. Dulness, and the absence of vocal fremitus, even should the breath sounds not be quite inaudible, often warrant the diagnosis.

When effusion is very copious the whole lung is compressed, the semi-circumference of the affected side is increased, and the intercostal spaces may be bulged. In addition, if the effusion be on the left side, the heart is displaced to the right, and may be seen pulsating below the right nipple, and the spleen is pushed downwards; if on the right side, the liver is pushed downwards, and the heart may be displaced somewhat to the left.

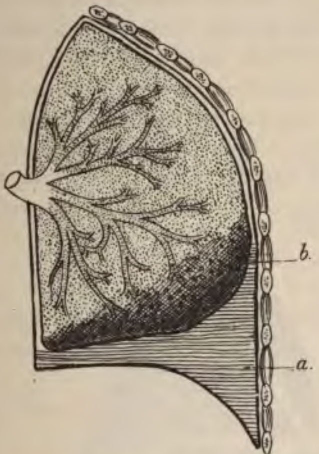


FIG. 62.—Showing at *a* moderate pleural effusion with, at *b*, bronchial breathing and crepitations over the compressed portion of lung.

When the whole lung is not compressed the breathing over the non-compressed part is puerile. When the greater part of the lung is compressed the breathing over the other lung is puerile.

Phthisis.

Phthisis usually begins at the apex of one or other lung.

In the early stage it is characterised by diminished expansion and perhaps flattening, dulness on percussion, breathing more or less bronchial in character, crepitations, and increased vocal resonance. The crepitations may only be present in the supra-clavicular and supra-spinous areas or just below the outer end of the clavicle, so the auscultation of these regions ought never to be neglected. When the crepitations are abundant and present during both inspiration and expiration, the type of the breathing may be quite obscured, and when this is so it ought to be mentioned.

In the more advanced stages, when the changes in the lung and pleura are of a very varied kind, the physical signs vary greatly. There are marked diminution in expansion, considerable flattening, and dulness on percussion, or if a large cavity be present at the apex, the cracked-pot sound may be elicited. Vocal fremitus is usually increased. The breath sounds are bronchial, cavernous, or amphoric. The accompaniments are coarse crepitations, often gurgling, metallic, or tinkling in character, and along with this often wheezing and squeaking sounds which belong to the type of rhonchi. The vocal resonance is bronchophonic, pectoriloquous, or amphoric.

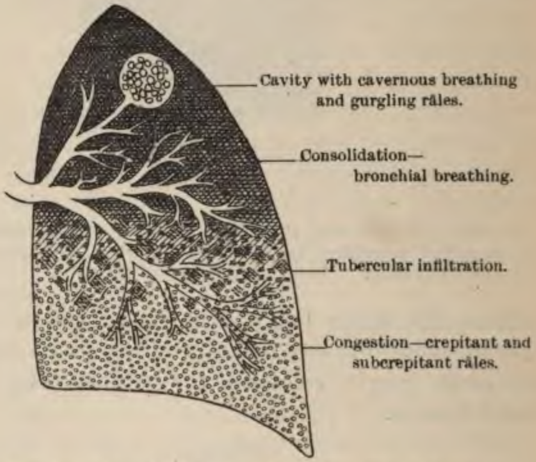

Cavity with cavernous breathing and gurgling râles.
Consolidation—bronchial breathing.
Tubercular infiltration.
Congestion—crepitant and subcrepitant râles.

FIG. 63.—Showing phthisis at various stages in one lung, the physical signs depending on the stage.

The sounds vary greatly at different times, owing to the variable state of the alveoli, the cavities, and the bronchi. If the bronchi are filled with secretion no air may enter the affected part and little sound of any kind may be audible. If cavities are filled with secretion and their communication with the bronchi occluded, the coarse gurgling crepitations which may have been present at one examination disappears, while if the patient be asked to cough the sounds may again be produced.

Pneumothorax.

Definition.—This term is applied to the presence of air in the pleural cavity.

Causes.—It most commonly occurs in cases of tubercular disease of the lungs, the pleura becoming undermined and perforated by the tubercular affection, so it can only occur where adhesions have not been formed between its visceral and parietal layers. It may also result from pyæmic abscesses of the lung bursting through the pleura; from gangrene of the lung involving the pleura; and from wounds of the lung, either by puncture though the thoracic wall, or by laceration from the end of a broken rib. It may also be caused by violent respiratory efforts, but when this happens it is almost certainly owing to a weakened condition of the part where rupture occurs. In a considerable number of cases it is impossible to be quite satisfied as to the precise cause, for it supervenes in individuals who are apparently free from pulmonary disease, and who have not been subjected to strain or injury.

Physical Signs.

Inspection may show distinct *bulging* of the affected side, or of part of the side. Bulging of part of the side occurs in cases where partial pleural adhesions exist. Where the pleural cavity is completely obliterated by adhesions air cannot, of course, escape into it, but air may escape into a part which is not obliterated, and when this occurs the bulging is local. In some cases, however, there is no bulging. *Absence of respiratory movement* over the affected part is always noted. There may be slight up and down movement, but this must be distinguished from the true inspiratory movement, which is one of expansion.

Percussion.—The percussion sound varies in different cases, the variation depending on the size of the air-containing space, the degree of tension of the air within the space, and the patency of the perforation between the lung and the pleural cavity. The aperture tends to become closed by the approximation of its edges, as a result of the collapse of the lung, and also from the effusion of lymph round and over it. An aperture which has in this way become temporarily closed often re-opens during a fit of coughing, from the violence of the expiratory act, and the increased intra-thoracic pressure which accompanies it. When the air in the pleura is under high pressure, and the aperture in the lung closed, the percussion sound is dull; when the aperture is patent, or the pressure not great, the percussion sound is clear, resonant, and may be distinctly tympanitic. In a considerable number of cases, however, the percussion sound is not sufficiently distinctive to be of much value. When the condition occurs on the left side, and causes displacement of the heart, we have noted that the tympanitic character was more distinctly marked over the normal cardiac region than elsewhere.

Vocal fremitus is absent.

Auscultation.—The auscultatory signs are very variable, but often present phenomena of great interest. The ordinary respiratory sounds are absent; in fact there may be an entire absence of any sound whatever when the ear is first applied. There may, however, be a sighing musical sound, resembling the sound made by the wind passing through a crevice. This may be present during inspiration or expiration, usually most markedly so during the latter, and it may continue after inspiration or expiration has ceased: the phenomenon is due to air passing through the aperture in the pleura, the aperture being in great part closed, a small chink only remaining open. If the patient be asked to cough various changes may be noted: a respiratory sound may become audible, and be distinctly amphoric in character. The sound of the cough also may have the amphoric character,—that is, it sounds as if the patient had coughed into a chamber containing air only, the sound, however, being not only hollow in character, but also having a very distinct and pleasing metallic character. Further, a phenomenon which is known as metallic tinkling may be present: this sound

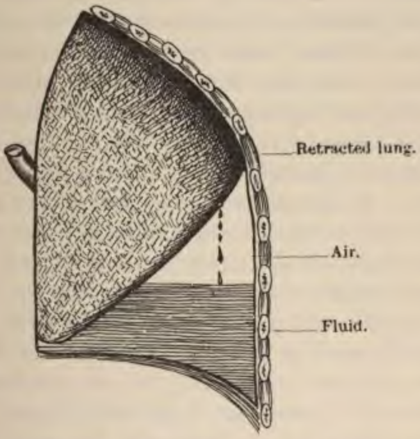


FIG. 64.—Showing the condition of parts in hydro-pneumothorax from a perforation in the pleura. Metallic tinkling is represented by drops falling on to the surface of the fluid.

exactly resembles the dropping of water on to the surface of fluid contained in a circumscribed space. It may be imitated by dropping water on to the surface of water in a water-bottle, and is often heard in house cisterns. This sound may be present when the patient is breathing quietly, but it may only be present after coughing. The number of tinkles also varies; a solitary one after coughing may be all that is audible. The phenomenon is probably only present when there is fluid as well as air in the pleural cavity: it is produced by drops falling from the walls of the cavity on to the fluid, or by the bursting of air bubbles on the surface of the fluid. The explanation is that the act of coughing more or less shakes up the fluid, and splashes it up the sides of the cavity from which it drops down; or the disturbance of the fluid leads to the formation of air bubbles, which burst.

Another of the metallic phenomena which is often present is elicited by listening through the stethoscope while percussion is exercised with two coins. The sound which is then heard is usually referred to as the *bell sound;* as a rule, however, it has much more what Dr. Wyllie has happily described as the *anvil sound*, for the sound has a very striking resemblance to that, heard at a distance, produced by striking with a hammer a smithy anvil. If the stethoscope be retained at a fixed point, and the coins be moved about, the limits of the air-containing space may be accurately defined, for as soon as the coins pass to a part of the chest which does not cover the air space the anvil sound is lost. This anvil sound cannot always be elicited, even in cases where there can be no doubt that pneumothorax is present; the precise meaning of this is somewhat obscure, but it probably depends upon the degree of tension in the air space, and the patency of the aperture in the pleura.

The various phenomena collectively termed metallic owe their metallic character, as has been already indicated, to the presence of an air-containing space; the sounds, however produced, acquiring the metallic character from the mode in which reverberation takes place in the cavity.

Vocal resonance may be absent, but it may be distinctly amphoric, and even have a metallic character or clang. This has been referred to under auscultation when indicating the phenomena produced by coughing, the resonance of the cough being the same in kind as the resonance of the speaking voice, although the amphoric character of the former may be elicited when the latter cannot.

Hippocratic succussion is the term generally applied to the splashing sound which can be produced by shaking the patient or by any sudden movement on his own part. It is really a splash, and can only be produced when both air and fluid are present. It bears the name of Hippocrates, as his is the earliest description of it extant.

When both fluid and air are present the condition is called *hydro-pneumothorax.* The presence of air in the pleural cavity is probably a factor in producing inflammation of the pleura, and accompanying it fluid is poured out. The fluid may be serous, sero-purulent, or purulent. It tends to become purulent, even if not so from the beginning, but its character can only be definitely settled by an exploratory puncture with a hypodermic syringe. When fluid is present its position can be mapped out by percussion and auscultation, or by a combination of these. The percussion sound over the fluid is dull, there is absence of all respiratory sound, and the anvil sound is lost when the coins reach the part of the chest wall covering the fluid. The position of the fluid is of course at the most dependent part of the cavity, and alters with the position of the patient.

Displacement of organs has been already referred to. When the condition is on the left side, the heart may be felt beating as far to the right as the right nipple. The mediastinum may also be displaced, a clear percussion sound being present as far as the right edge of the sternum. When the lesion is on the right side the liver may be pushed down, the heart displaced somewhat to the left, and the mediastinum also to the left. Dyspnœa is most distressing when the lesion is on the left side, the action of the heart then being more embarrassed. On whichever side the lesion exists the breathing power of the affected lung is annulled, and the circulation in it is very greatly impeded. We believe we have noted that the pouring out of fluid has considerably increased the dyspnœa, and that it has been relieved somewhat by its removal.

SPUTA.

The sputum presents some important naked-eye characters, and in some cases requires to be examined microscopically.

It may consist of materials derived from the mouth, nares, or fauces, when its composition is not as a rule of much importance. The possibility of its origin in these situations has to be remembered, for patients not infrequently complain of spitting blood, a symptom popularly associated with serious lung disease, and therefore alarming, but which on examination may be found to proceed from the fauces or posterior nares. It is sputum from the respiratory tract which alone requires special consideration here.

Naked-eye characters.—In persons affected with a slight chronic catarrh, evidenced by the presence of a little cough, and most frequently present in the morning, the attack of coughing often terminates by the expectoration of a piece of toughish mucus of a *greyish or blackish grey colour.* The colour in these cases is due to the presence of dust which has been inhaled. The condition is not, as a rule, of much clinical significance.

In acute bronchitis the expectoration is abundant, more or less viscid and *frothy.* It consists of the secretion from the inflamed mucous membrane of the bronchi, and its frothy character is due to its admixture with air. In œdema of the lungs the secretion is somewhat the same, but it is more watery, and much more profuse in quantity. In chronic bronchitis, in addition to the foregoing characters, it contains masses of more tenacious expectoration of a yellow colour, the colour being due to the presence of pus cells, and indicating a further stage of the inflammatory process. When very profuse it is known as *bronchorrhœa.*

In croupous pneumonia the sputum is described as *rusty* or *prune-juice* sputum. Its reddish-brown colour is due to the presence of red blood corpuscles in it.

In phthisis the expectoration is usually very profuse ; it is always purulent in character, and is often expectorated in tenacious lumps or masses, which are known as *numula,* the sputa being known as numulated. It is secreted from the walls of cavities and from the bronchi. It may be streaked or coloured with blood. Occasionally the expectoration is purely *sanguineous,* and then it is usually bright red, and may be frothy from its admixture with air.

In bronchiectasis there is often a periodic discharge of a great quantity of expectoration, the result of the emptying of a bronchiectatic cavity : it usually has a fœtid odour from decomposition.

In gangrene of the lung the expectoration may be more or less blood-stained, and it always processes an extremely offensive and penetrating odour.

It not infrequently happens that a purulent expectoration has its origin in an empyema which has perforated the lung and is discharging by the bronchi. The sudden expectoration of a considerable quantity of pus points to this, or to a mediastinal abscess having burst into a bronchus, but it may also have its origin in an abscess of the pulmonary parenchyma, which has formed an opening into a bronchus.

Microscopical Examination.—The sputum is examined microscopically for elastic fibres and for micro-organisms.

To examine for elastic fibres, part of the sputum requires to be boiled with caustic potash, then allowed to settle in a conical glass, and the sediment examined. They are found in cases of phthisis, and are derived from the seat of destructive change in the lung.

Of micro-organisms the most important is the *Bacillus tuberculosis.* The following methods may be followed to demonstrate it. First, a very convenient and simple method is by means of Heneage Gibbs' double stain: a thin layer of sputum is spread between two cover-glasses, these are separated, and the film of sputum dried by passing through the flame of a spirit lamp. A little of the stain is poured into a watch-glass, which should be held over a spirit lamp until steam rises from it, then the cover-glass is put into it with the film downwards and left for five minutes; it is then lifted out with a forceps, washed in water, and then in methylated spirit, until all the stain that will wash off has been removed. After being dried, it is placed on a drop of balsam on a slide and examined. The bacilli are visible as small red bodies, while the rest of the field shows a faint blue colour. If a positive result be obtained by this method, it may be regarded as conclusive; but if the result be negative, it may be desirable to check it by the following:—The cover-glasses, having a film spread on them as described above, are placed in a solution of fuchsin for five or ten minutes if heated, or two or three hours if cold; the cover-glass is then dipped for a few seconds in diluted nitric acid, one in three or four; this decolorises everything except the bacilli: stain for a few minutes in a dilute solution of methylene-blue, wash in distilled water, dry, and mount in Canada balsam dissolved in turpentine or xylol on a slide. The bacilli appear red, the cells, &c. blue.

CHAPTER VII.

THE ALIMENTARY SYSTEM, INCLUDING THE ABDOMINAL VISCERA.

THE MOUTH.

The Teeth.—The state of the teeth is a matter of considerable practical importance, as a moderate number of good teeth is essential to efficient mastication, and imperfect mastication is one of the commonest faults in persons who suffer from various forms of indigestion.

The Gums present characteristic appearances in some diseases. In lead poisoning they show a blue line at their junction with the teeth. In hæmorrhagic purpura and scurvy they are spongy and bleeding, or bleed on slight pressure. In anæmia they are pale and bloodless. In poisoning from mercury and other metals, and in syphilis, they may be ulcerated.

The Mucous Membrane of the cheeks and hard palate only exceptionally presents appearances of importance. In the eruptive fevers the rash characteristic of the special fever is usually present on it. In children, and in adults suffering from exhaustive diseases, aphthous patches may be present both on the cheeks, palate, and gums.

The Tongue.—The tongue has from time immemorial been scrutinised by the practitioner of medicine, and its appearances have been regarded either as an indication of the general condition of the patient, or simply as an indication of the condition of the digestive organs, and more especially of the stomach and liver.

Leaving out of account the condition of the voluntary movements, which will be referred to more particularly under the nervous system, the points to note are as follows :—(*a.*) *Tremulousness.*—This may be the result of temporary nervousness, or due to organic changes in the central nervous system, or it may be part of a general condition, as in alcoholism and the acute febrile disorders. (*b.*) *Size and shape.*—Some tongues are large and flabby, with a broad tip ; others are firmer, and protruded with a pointed tip. The former often presents an indented edge, produced by pressure against the teeth. (*c.*) *Humidity.*—The tongue may be dry, moist, or watery. In health the tongue is moist ; it is watery in certain forms of indigestion ; and it is dry in arrest of the salivary secretion, and in acute diseases. (*d.*) *Condition of its covering.*—The appearances presented by the mucous membrane within the limits of health vary considerably in different individuals. The term *furred* is applied to the tongue when its surface looks as if it consisted of a fine fur. It may be whitish or brownish in colour ; when the latter, it is frequently confined to the posterior part of the organ. A whitish fur is often diffused over the whole dorsum. When the papillæ project as red points through the fur, the tongue is known as a *strawberry tongue*, from its supposed resemblance to that fruit. The tongue is *coated* when there is a thick covering over whole or part of it, which may be whitish, yellowish-brown, or even black in colour. In addition a coated tongue may be moist or dry—the combined dark and dry coating or encrustation only occurring in severe forms of constitutional disturbance. In some cases after the coating comes off the tongue is red, bare, and raw-looking ; in others, in which there has been no antecedent coating, the tongue has a similar red and raw-looking appearance. (*e.*) *Abrasions.*—The tongue further may be the seat of cracks and fissures, these being the result in some cases of prolonged indigestion, but, when present in acute diseases, the result of the dryness of the organ combined with other causes.

THE PHARYNX.

Few words need be spent upon a method of examination which is practised in the domestic circle as well as by physicians. In order to obtain a view of the pharynx the patient is seated before a window, better still in such a position that a strong light can be thrown into the mouth by means of a laryngeal reflector. The tongue is then depressed either with the handle of a spoon or an instrument specially constructed for the purpose, called a tongue depressor. A view is thus obtained of the anterior pillars of the fauces, the soft palate, uvula, and tonsils, while in the background is seen the posterior wall of the pharynx. It must be particularly noted whether the parts seen are normal in colour and size; the mobility of the soft palate should also be tested by making the patient phonate. The presence of mucus in excessive quantity will at once make itself apparent should this condition exist, while sometimes the pharyngeal secretion shows a great tendency to dry up into more or less viscid crusts as soon as it is pressed out. We need not here refer further to the occasional presence of false membrane (as seen in diphtheria), masses of leptothrix, &c.

A better view of the pharynx in its deeper portions may be obtained, as pointed out by Voltolini, by making the patient protrude the tongue, grasping it, and then introducing the tongue depressor. If at the same time the thyroid cartilage be pressed up and the patient be made to retch, the whole epiglottis is often exposed to view. The act of retching further enables us to see the folds of mucous membrane which lie behind and parallel to the posterior pillars of the fauces, and are often hypertrophied or swollen.

In young children who refuse to open the mouth the pharynx can often be examined by having their hands fixed, then compressing the nostrils and introducing the tongue depressor, when want of air compels the patient to separate the teeth or jaws.

As a matter of course, palpation with the finger, or even probing, may be necessary in certain cases of pharyngeal disease, but these methods are rarely required. If the parts be unduly irritable—as sometimes happens in those who live freely or are of a gouty habit—it may be advisable to blunt the sensibility of the pharynx with cocaïne before examination.

THE ŒSOPHAGUS.

The presence or position of strictures has to be determined by means of œsophageal bougies, and their mode of use hardly falls to be considered here.

THE ABDOMEN.

Regions.

For convenience of description the abdomen is divided, as represented in Fig. 42 (*ante*), or, as in the following figure, by

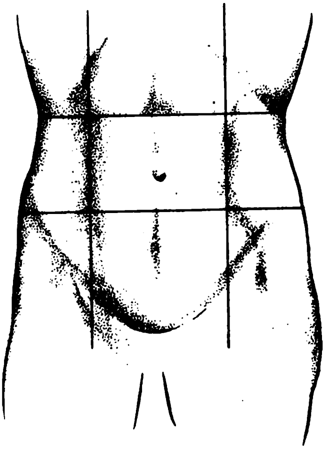


FIG. 65.—Showing the abdominal regions.

two vertical and two transverse lines. The vertical lines are drawn upwards from the middle of Poupart's ligament, one on each side; while the transverse are drawn one at the level of the lowest part of the thorax, the second at the level of the highest point of the iliac crest. The regions thus marked out are represented in the diagram, and are from above downwards as follows:—

Right Hypochondrium.	Epigastrium.	Left Hypochondrium.
Do. Lumbar.	Umbilical.	Do. Lumbar.
Do. Iliac.	Hypogastrium.	Do. Iliac.

The structures occupying these regions are enumerated in the ordinary text-books on anatomy.

General Examination.

Inspection.—By inspection the shape, the degree of distention, and the state of nutrition are observed. If there are local bulgings their position is to be specified. The presence and position of pulsations are to be noted. The measurement of the circumference of the abdomen is taken at the level of the umbilicus. A record of the measurements taken at fixed periods is necessary in cases of ascites, &c.

Distention is due either to the accumulation of gas in the intestines, to fluid in the peritoneal cavity, or to tumour. When due to the first of these, the abdominal wall is distended forwards, the distention being greater in the umbilical region; when to the second, the distention is most marked in the flanks, but if there is much fluid present there is distention forwards as well as laterally. This description is applicable mainly to the appearances in the recumbent position; when the sitting or the erect posture is assumed the lower part of the abdomen bulges, from the fact that the fluid changes with the change of position, and always gravitates to the most dependent part.

The upper part of the abdomen may be bulged by a distended stomach, or by an enlarged liver or spleen, but these will be more fully dealt with under their respective sections.

In the lower part of the abdomen the gravid uterus, uterine or ovarian tumours, and a distended bladder lead to local bulging. Large uterine and ovarian tumours lead to distention of the whole abdomen.

Laterally enlargements of the kidney may produce bulging.

Umbilical hernia, when present, is to be noted; it occurs not infrequently as a result of distention from ascites.

The epigastric veins may be unduly prominent from interference with the portal circulation, or from obstruction to the inferior vena cava.

In women who have borne children, or in whom the abdomen has been greatly distended from any other cause, there are white lines (*lineæ albæ*) of scar tissue on the abdomen and upper part of the thighs.

On the other hand a drawn-in condition or *retraction* of the upper part of the abdomen is a marked feature in meningitis, especially in children.

Palpation.—After inspection, the abdomen is palpated. To facilitate this proceeding the patient should lie flat on the back with the knees drawn up, and be requested to continue breathing quietly. This last point is of importance, for there is a tendency to hold the breath, and when this is done the abdominal muscles become tense and resisting; it also takes the patient's mind away from the abdomen, and concentrates it on respiration.

The condition of the abdominal wall as regards the amount of subcutaneous fat, the presence of œdema, the lax or tense state of the muscles, is to be noted.

The characters of local bulgings are then investigated. Those due to affections of the abdominal wall have to be distinguished from those caused by intra-abdominal conditions. When due to the latter, they are usually elastic or hard, and are caused by enlargement of a solid organ, or a tumour, and the surface may be smooth, or nodulated, or umbilicated. The tumour may be fixed, or it may be moveable, or it may move with respiration. Moveable tumours are not very common, one of the commonest being loose or floating kidney. Tumours which move with respiration are usually connected with the liver or spleen, for these organs move with the diaphragm during respiration, and tumours connected with them necessarily participate in their movements. This is an important diagnostic point. The movement may be visible, at other times it can only be made out by placing the hand, especially the fingers, flat and firmly on the part; and it is necessary to be sure that the diaphragm is itself moving freely, for patients are prone to impede its action when the abdomen is palpated, and if its action is arrested during examination the observations made will not be reliable.

The presence of fluid, as ascertained by palpation, is fully described under ascites.

The limits and characters of pulsations are also to be noted.

Percussion.—The greater part of the abdomen is normally tympanitic, but if the intestine be filled with solid matter or with fluid at any part the sound is dull: this is specially noticeable over the great intestine when it is loaded with fæces. The difference in pitch over the stomach, large intestine, and small intestine respectively can sometimes be distinguished, but it requires much practice, and is surrounded by many possibilities of error. The percussion sound is also dull over fluid, over the solid organs, as the liver and spleen, over a distended uterus or bladder, and over all abdominal tumours, if not overlaid by intestine. The tympanitic percussion sound over the stomach and intestine is raised in pitch when tightly distended with gas.

We have now to deal more in detail with the physical examination of some special morbid conditions of the abdominal cavity and of the various abdominal organs.

Ascites.

By this term is meant accumulation of fluid in the abdominal cavity.

Physical Examination.

Inspection.—The abdomen is distended, the degree varying with the quantity of fluid. When the quantity is small the anterior surface is normal or slightly rounded, and there is lateral bulging in the flanks. If the patient be lying inclined to one side the bulging is greater on the side towards which he is inclined; if he be in the sitting or standing posture the bulging is greatest at the lower part of the abdomen. When there is much fluid the roundness of the anterior aspect, and also the bulging of the flanks, are greater; the upper part, including the epigastric and both hypochondriac regions, is also distended, even the lower ribs being pushed outwards; in short the whole abdomen becomes barrel-shaped and the abdominal wall exceedingly tense. In addition, the umbilicus is stretched, and often projects beyond the surrounding surface. Umbilical hernia not infrequently follows upon ascites, especially if there is much cough, for the right and left rectus abdominis muscles are separated by the abdominal distention, and coughing favours protrusion in this situation, as it is one of the weak points in the abdominal wall. When there is much distention the pressure on the inferior vena cava and the iliac veins leads to congestion of the veins of the lower extremities, and as a consequence to œdema of them.

Percussion.—When the patient is on his back the percussion sound over the anterior aspect is clear and tympanitic, as the intestines float on the fluid and are in contact with the abdominal wall in this situation. In extreme degrees of distention the clear percussion sound is only present above the umbilicus, owing to the intestines being pushed up by the fluid, while above this the liver dulness reaches a higher point than normal, the thorax being encroached on by the upward pressure of the intestines, liver, and diaphragm. The flanks, the lateral parts, including the hypochondriac, lumbar, and iliac regions, and the hypogastrium, are dull.

This is due to the fluid gravitating to the posterior, lateral, and lower parts of the cavity, the percussion sound over fluid always being dull. If the patient be turned on the side the level of dulness on the dependent side rises, that is, it approaches nearer the middle line, while a tympanitic, or at least a clearer, sound is present over the elevated side: this is due to the fluid being free in the abdominal cavity, and changing with the position of the trunk. A similar phenomenon is produced by moving the patient from the recumbent to the sitting posture, the limit of dulness in the hypograstrium then rising higher, that is, towards the umbilicus.

While the foregoing is the result of percussion in the majority of instances, cases are frequently seen in which, notwithstanding the presence of considerable ascites, there is a tympanitic sound on percussion in one or other flank: this is to be explained by gaseous distention of the colon bringing it into contact with the abdominal wall at the part where the tympanitic sound is obtained.

Palpation.—The abdomen has an elastic feeling. If one hand be placed flat over one lumbar region, while the opposite lumbar region is tapped with the fingers of the other hand, a distinct wave-like impulse is felt when much fluid is present; if, however, the quantity of fluid be small, this is not obtained, but it can be elicited by a similar proceeding applied to the lateral aspect by, for instance, placing one hand over the iliac region while the lumbar region of the same side is tapped with one finger of the other hand. The impulse may be caused either by the fluid or by the intestine floating on it, but it can only be produced when fluid is present. A true wave is not obtained in flatulent distention.

Conditions which produce ascites.—Ascites is caused by valvular lesions of the heart, more especially mitral stenosis and incompetence, with secondary tricuspid incompetence and general venous engorgement; by diseases of the lungs interfering with the circulation through them and producing tricuspid incompetence and general venous congestion; and by kidney disease, more especially the chronic parenchymatous form. In addition, there are conditions which act directly on the portal circulation and obstruct it: these are obstruction (*a.*) *within* the liver from cirrhosis or tumours; (*b.*) *without* the liver from the pressure of a tumour or from phlebitis of the trunk of the vein. In all these, with the exception of nephritis, the pouring out of the fluid is the result of venous congestion, and is a purely passive process.

Fluid may also be present in the abdominal cavity as the result of inflammation, either simple or tubercular, or as the result of irritation from diffuse malignant disease. The fluid may be purulent or coloured with blood, and if jaundice be present it is bile-stained.

If the fluid contain much blood it is probably the result of ulceration of a malignant nodule, or of hæmorrhage from extremely distended capillaries adjoining or overlying malignant nodules.

The fluid is milky when the transudation results from obstruction to the thoracic duct. This condition is known as chylous ascites.

Peritonitis.

Peritonitis is inflammation of the peritoneum. In addition to pain and general constitutional disturbance, there are physical indications of its presence. The patient lies on the back with his legs drawn up, in order to relax the abdominal parietes as much as possible. Respiration is almost entirely if not altogether thoracic, as any movement of the diaphragm increases the pain by the movement communicated to the abdominal viscera. The abdomen is distended by the accumulation of gas in the intestines. This is known as *meteorism*, and results from paralysis of the muscular coat of the intestine, consequent on the inflammatory action. A further result of the paralysis is constipation. The abdomen, as might be anticipated, is tympanitic, but percussion must not be used more than is absolutely necessary, as there is extreme tenderness. In severe cases there is effusion of fluid, and if it is present in any quantity the physical signs indicative of its presence may be obtained; it does not, however, so readily change its position as in ascites from more mechanical causes, owing to the gluing together of the coils of the intestines to one another and to the parietal peritoneum. In the form of peritonitis known as *tabes mesenterica* the enlarged abdomen has a doughy feel, and the enlarged glands may be felt.

THE STOMACH.

Anatomical relations.—The stomach, which is flask or pear-shaped, occupies part of the left hypochondrium and the epigastrium. It is situated below the diaphragm and the liver, and above the transverse colon. A small part of its anterior surface is frequently in contact with the anterior abdominal wall: this is known as *Traube's area,* and lies to the left of the mesial line, being bounded on the left by the edges of the cartilages of the asternal ribs, and superiorly by the anterior edge of the left lobe of the liver.

Its *posterior anatomical relations* are as follows:—The *cardiac orifice* is placed deeply, and is in front and to the left of the body of the twelfth dorsal vertebra. The *pylorus* is in the middle line and opposite the eleventh and twelfth dorsal and first lumbar vertebræ. When distended it may be moved three inches to the right of the mesial plane.

Anterior relations.—The *cardiac* orifice is at the level of the space between the tip of the ensiform and the seventh left cartilage. The *pyloric* orifice is in the mesial plane, or a finger's-breadth to the right of it, at the level of the junction of the middle and lower thirds of a line drawn from the sternum to the umbilicus. Its position is shown in Frontispiece I.

The superior limit of the *fundus* of the organ is on a higher level than the cardiac orifice. The frontispiece represents the stomach smaller than it is represented in most books; and the average of observations in the *post-mortem* room show it to be larger, but we are distinctly of opinion that the average there is abnormally large, that in fact the majority of stomachs, as seen on the *post-mortem* table, are dilated; the frontispiece has therefore been made to represent the organ nearer what we believe to be the normal size.

Inspection.—In the normal state inspection gives no information as to the state of this viscus. If, however, it be greatly distended, it leads to bulging of the abdominal wall, and its outline may be traced by the eye.

Percussion.—In the majority of individuals, and when the stomach is not distended with food, a tympanitic sound may be elicited in what has been referred to as Traube's area. When the fundus is dilated with gas the area over which the tympanitic sound can be produced is extended. The direction in which this extension takes place is upwards and outwards, more especially upwards. In even moderate degrees of dilatation it will be found that, if percussion be performed over the heart, and continued downwards just within a vertical line drawn from the apex, the percussion sound changes suddenly from the dull sound produced by the heart to a distinctly tympanitic sound, which is at first somewhat muffled, but which, as we proceed downwards, loses this muffled character. Percussion must be moderately strong for this. The upper limit of the muffled tympanicity marks the upper limit of the fundus. The muffling of the sound results from the diaphragm, and the liver or lung intervening between the organ and the surface. Where the viscus is no longer covered by these structures the sound becomes clearly tympanitic. By percussing to the left, the outer limit of the great curvature can often readily be defined. If, however, the colon be distended with gas, the delimitation of this border is more difficult, but may be made out by noting a change in the pitch of the sound as we pass from stomach to colon. From this it will be understood that the dilated fundus lies under the costal cartilages and the ribs, below and to the left of the cardiac dulness, as represented in Fig. 68. The precise area over which tympanicity may be elicited varies of course with the degree of dilatation, but

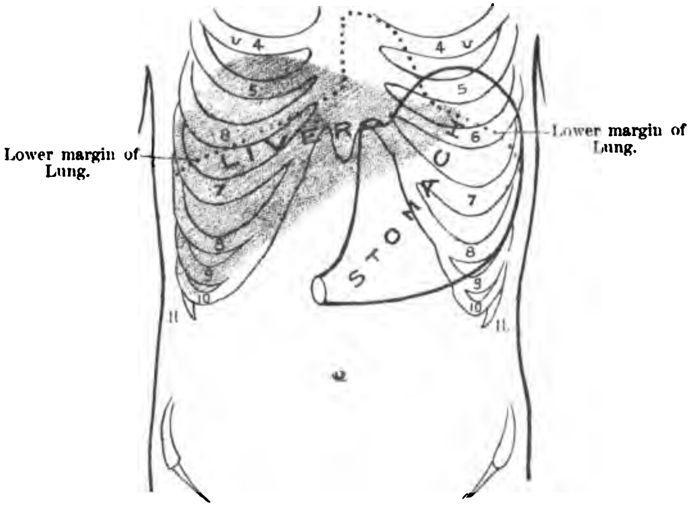


FIG. 66.—Showing a moderately distended stomach.

in our experience the presence of tympanicity over any part of the cartilages or ribs adjoining Traube's space is an indication of a degree of flatulent distention of the fundus. This condition is very commonly met with in persons who do not suffer acutely from gastric disturbance, but who on inquiry will be found to be subject to flatulence, and not infrequently to acidity and even pain. The recognition of these minor degrees of dilatation is of considerable clinical value, and has been largely overlooked or neglected. The determination of the inferior border of the stomach is surrounded with greater difficulty, and it is often impossible to satisfy one's-self as to its position; this is owing to the frequent distention of the transverse colon with gas, giving a percussion sound which cannot be distinguished from the sound over the stomach. When the transverse colon is distended with fæces or empty this difficulty is not present. In some cases a difference in the pitch or quality of the note indicates that we have passed from the stomach to the colon. Another source of error is that the stomach when distended frequently contains a large quantity of fluid, and this gravitating to the most dependent part, gives a dull note before we have left the organ.

It may be so enlarged that it occupies a great part of the abdominal cavity, reaching even as low as the pubes. Its condition, however, varies. It may be blown up like an inflated bladder, and project forwards when the abdomen is opened, or it may not be distended to anything like this degree, but, instead, be stretched and dragged downwards. This latter condition is illustrated in the following diagram, taken from a case in the *post-mortem* room, in which, as will be seen, the great curvature of the stomach was as low as the pubes. Owing to the practical fixity of the cardiac orifice, and comparative fixity of the pylorus, when the viscus becomes stretched in its longitudinal diameter, without being distended in circumference, it assumes the U shape seen in the diagram. The diagram represents an extreme case, but minor degrees of the same type are commonly seen.

This latter condition is described as *dislocation of the stomach.* It is not capable of recognition by percussion, but the production of the splashing sound in it by palpation would warrant the diagnosis. The transverse colon is displaced with the stomach; in the case from which the diagram was taken it was in the pelvis.

Palpation.—When the stomach is the seat of ulceration or malignant growth there may be pain or tenderness on pressure; this, however, always leads to a tense state of the abdominal muscles, which hinders a satisfactory examination being made. Malignant growths in the stomach can hardly ever be felt. Malignant affection of the pylorus can, however, at times be distinctly felt; it is known by its anatomical position, and by the signs of obstruction present.

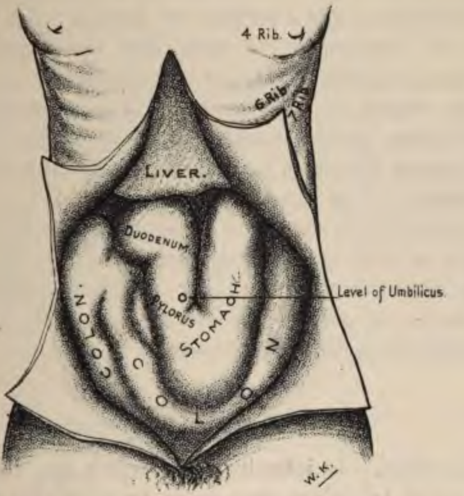


FIG. 67.—Taken from the *post-mortem* room, showing the U shape often assumed by the enlarged stomach.

Evidence of dilatation of the stomach is obtained by producing a splashing sound in it. This is done by placing both hands on the abdomen over the organ as if to palpate it, pressing somewhat firmly inwards, and making a movement as if shaking the organ from side to side; this agitates any fluid which may be present in it, and if gas is also present a splashing sound is obtained. This sound can often be produced by the voluntary efforts of the patient either by suddenly drawing in the epigastrium or by suddenly jerking the diaphragm downwards. We have known the voluntary performance of these acts, repeated several times in succession, lead to the eructation of gas, and the disappearance of the phenomenon.

The sound is analogous to the succussion sound produced by shaking a patient when fluid and air are present in the pleural cavity.

The precise limits of a dilated stomach can be defined by introducing first a solution of tartaric acid, then a solution of bicarbonate of soda into the organ, but this is hardly a desirable method of investigation.

The causes of dilatation of the stomach are pyloric obstruction, gastric catarrh, and atony of the muscular coat. A degree of dilatation is common in cases of general debility, irrespective of its immediate cause.

Diminution in the size of the stomach occurs to a marked degree in obstruction at the cardiac orifice, the most common cause of stricture being malignant disease. It also occurs in old and debilitated people, and in some long-standing and exhausting diseases.

THE SMALL INTESTINE.

Inspection.—In intestinal obstruction gas accumulates in the small intestines, and leads to great distention of the abdomen, and if the abdominal wall be thin the position of the coils of intestine is shown on it in somewhat transverse ridges and furrows. When this appearance is present it often denotes that the stricture is at the cæcum or lower part of the ileum. The degree of distention may sometimes help to indicate roughly the position of the stricture, for the nearer the stricture is to the duodenum the less is the distention. The vermicular movement of the intestine may also be visible through the emaciated parietes.

Palpation.—In thin subjects hard fæcal lumps are readily felt in the small intestine; they must not be mistaken for tumours.

Percussion.—In the normal condition the percussion sound is tympanitic; when greatly distended with gas, as in peritonitis and obstruction, the sound is lower in pitch but retains its tympanitic character.

The rumbling sounds produced in the intestines are known as *borborygmi*, and are caused by the passage of gas and semi-fluid matters.

THE COLON.

The position of the various sections of the colon is represented in Frontispiece I. It is necessary to bear in mind that the posterior aspect of the ascending and descending portions are not covered with peritoneum, and are united to the posterior abdominal wall by loose connective tissue. This is important, as purulent inflammation set up, say, at the cæcum may spread upwards in this tissue without implicating the peritoneum, while on the other hand a purulent inflammation beginning in the loose connective tissue round the kidney may extend downwards behind the colon, and reach the cæcum or sigmoid flexure.

Owing to the position of the transverse colon immediately inferior to the stomach, distention of it with gas gives a percussion sound which often cannot be distinguished from that of the stomach. Not infrequently the colon is distended when the stomach is unusually small, and when this is the case its percussion sound is almost certain to be mistaken for that of the stomach. In some cases, however, a difference in the pitch or in the amplitude of the percussion sound between the two structures may be made out. While the differential percussion of stomach and transverse colon is thus surrounded by possibilities of error, we may by repeated examination satisfy ourselves as to the condition of the stomach. Perhaps the most conclusive proof that it is the stomach with which we are dealing, is to elicit the splashing sound referred to under the examination of that viscus.

Malignant stricture of the colon may occur at any point, but its most common sites are the cæcum, the hepatic, splenic, or sigmoid flexures. In all these the distention of the part above the stricture aids the diagnosis, and can be made out by palpation and percussion. The physical examination of the colon is of great value in other cases, for patients are often found to have one of its sections, most commonly perhaps the ascending portion, loaded with fæces, and if the condition is not recognised we miss giving them the relief they seek. The outline of the distended ascending transverse or descending colon can be made out with sufficient definiteness by means of palpation and percussion, but it must not be expected that it has the hardness and firmness either of a solid tumour or of an enlarged liver; it is more doughy in feeling than either of these. When distended it pushes the small intestine aside, and comes much more to the front than in the normal condition.

The cæcum, with the vermiform appendix, is a part of the colon which requires special reference, as it is peculiarly liable to inflammatory attacks. The condition is known as typhlitis or perityphlitis, the two usually, although not invariably, being associated.

It is situated in the right iliac region and somewhat superficially. Inflammation is either the result of accumulation of fæces in it, or of ulceration of the vermiform appendix. As a consequence there is in the former dulness on percussion, and the sensation to the hand of fulness, resistance, or doughiness which have been already referred to as indicative of a distended colon. There are in addition the pain and tenderness which characterise inflammations; constipation, the result of paralysis of the muscular coat; and vomiting, which is present in all intestinal obstructions and some intestinal inflammations. Owing to the obstruction the small intestine becomes distended with gas, a condition known as meteorism. If abscess result, fluctuation may be made out, if it is in front; if, however, it is situated behind, it spreads upwards along the ascending colon in the connective tissue already referred to, and can hardly be made out by palpation; it is one of the conditions in which free exploratory puncture with the aspirator is required for diagnosis.

If ulceration of the appendix leads to perforation, peritonitis is set up.

A gurgling sound can be elicited in the cæcum by palpation in typhoid fever, but also in other and less important conditions: it simply denotes the presence of fluid or semi-fluid matter and gas in the part.

The Anus and Rectum.—The anus and rectum have often to be examined; the former for piles, fissures, fistulæ, condylomata, &c. The rectum is frequently the seat of cancer, and in all conditions where there are complaints specially referred to the lower bowel, it is desirable to make a digital examination; more especially is this necessary if treatment has not been successful in removing the symptoms complained of. Malignant stricture or malignant growth is frequently within reach of the finger. Another condition which we have seen producing extreme discomfort is the presence of a large and rounded scybalous mass in the rectum coming down whenever the patient went to stool, and in which the history of diarrhœa and straining were extremely misleading. Such a mass must be broken down by the finger, and its hardness makes this no very easy or agreeable task.

The sigmoid flexure is situated in the left iliac region: accumulation of fæces in it is therefore within easy reach of palpation. When stricture occurs below it, it becomes enormously distended with fæces or gas; we have seen it so greatly distended as to occupy the lower part of the abdomen stretching across to the opposite side, and even occupying the right iliac region.

THE ABDOMINAL LYMPHATIC GLANDS.

In some cases of malignant disease of the abdominal organs, the enlarged mesenteric and retro-peritoneal glands are felt and aid in forming the diagnosis. In tubercular peritonitis the enlarged glands may also be felt. They are also enlarged in cases of diffuse strumous disease of glands without being necessarily accompanied by peritonitis; also in lymphadenoma. The differential diagnosis between these two latter conditions can sometimes be made out only by careful physical examination of the lungs, and a consideration of the history and progress of the case.

THE LIVER.

Anatomical Relations.—The anatomical position of the liver is represented in the Frontispieces. Its superior surface is in contact with the diaphragm from before backwards. Its anterior, posterior, and lateral aspects are in contact with the ribs and the abdominal wall. Superiorly, where it lies under the right vault of the diaphragm, it reaches the level of the fourth space or lower border of the fourth rib. Its inferior limit practically coincides with the lower edge of the ribs, except in the epigastrium, where it crosses to the left about midway between the base of the xiphoid process and the umbilicus, but the precise position of this part of the lower edge varies greatly in different individuals. The organ in its upper part has the lung interposed between it and the thoracic wall: it is, however, uncovered by lung, and comes into immediate contact with the parietes, where the lung ends, and in the mesial line, at the base of the xiphoid process. Its position is therefore as follows:—

Median line.	Mammary and parasternal line.	Axillary line.	Posteriorly.
Base of xiphoid.	Sixth rib.	Eighth rib.	Tenth rib.

Its exact position varies with respiration, descending with the descent of the diaphragm and receding with its ascent.

It will aid in the comprehension of the position of this organ and its variations if it be remembered that, as Symington has pointed out, and as is seen in the Frontispiece and in Fig. 10, it is wedge-shaped, and that the long edge of the wedge runs along the lower margin of the ribs on the right side, and leaves it to cross the epigastrium obliquely.

Inspection.—Great enlargement of the liver leads to fulness and projection of the ribs covering it, and of the adjoining part of the abdomen. The projection of large malignant nodules, a hydatid cyst, or an abscess may also be visible.

Palpation.—In the normal condition the edge of the liver cannot, as a rule, be felt: this is true even of palpation in the epigastrium, owing to the thinness of its edge, and the little sense of resistance consequently given by it. When, however, the organ is enlarged the part of its surface in contact with the abdominal wall and its anterior edge can be readily felt. It can also be felt when it is displaced downwards by the pressure of effusion into the right pleural cavity; and in pronounced cases of emphysema, the emphysematous lung pushing both the diaphragm and it downwards. When enlarged the surface may be smooth or nodulated. If the enlargement is due to congestion, fatty infiltration, or waxy degeneration, the surface is smooth; when to malignant disease it is usually more or less nodular, and in some cases the umbilication of the nodules is felt. Over a hydatid cyst or an abscess there is fluctuation, or a feeling of elasticity. When the abdomen is distended, but not tensely, by fluid or gas, the anterior surface of the liver, if enlarged, may be felt by placing the fingers firmly over it and then suddenly pressing inwards: by this means the fingers are brought into sudden contact with the hard and firm organ, and we are conscious of the difference between it and the softer parts overlying it. The edge of the enlarged organ may be felt under similar conditions by pressing the fingers first inwards and then suddenly upwards: this may be done with either hand, the observer standing opposite the thorax of the patient.

Percussion.—Percussion of the liver gives two classes of results,—deep or comparative, and superficial or absolute dulness.

In finding the *deep dulness* percussion is begun in the second or third right intercostal space in the mammary line. We begin at that point to be sure of getting a good lung sound for purposes of comparison; we then percuss downwards, space by space, until the *slightest difference* in the character or the pitch of the sound is observed, and this point indicates the position of the uppermost part of the right lobe as it lies under the vault of the diaphragm and behind a considerable thickness of lung tissue. If greater precision be required, the two ribs above this space may be percussed to see whether the exact line be the space, or the rib above it. Here, as in the percussion of the lungs, ribs must not be compared with interspaces. The difference in the sound is slight, and is therefore called *comparative* dulness, but its accuracy may be relied upon, for we are in the habit of frequently checking this on the *post-mortem* table. This upper limit is almost invariably placed at too low a level. Percussion requires to be strong, as in the percussion of all solid structures lying behind air-containing organs. If desired, the same procedure may be followed on the lateral and posterior aspects of the thorax; but this is not as a rule done, although in some cases, especially in hydatid tumours or abscesses developing from its superior surface, it is of great service, as it indicates the presence of these projections, while they are beyond the reach of palpation. To find the inferior limit, percussion is begun in the nipple line, sufficiently far down in the abdomen to ensure getting a tympanitic sound, and continued upwards until a very slight difference is observed: this marks the lower edge. Percussion in this case must be *very light*, as the lower edge of the organ is thin and lies over air-containing intestine, and if not exceedingly light the sound from the intestine is brought out and quite masks the dulness of the liver. The measurement between these two points is that which is usually given as the *vertical liver dulness.*

The *superficial* or *absolute dulness* is taken from the point where the lung ends, so that what is really necessary in this case is to find the inferior margin of the right lung in the mammary line. This is done by percussing from above downwards, with extreme lightness, until all trace of clearness disappears, and the sound is absolutely dull even to light percussion. Now it will be found that from the point where the percussion sound changes slightly to the point at which it becomes absolutely dull, the sound presents several degrees of dulness, but if the percussion is sufficiently light, there need be no hesitation in proceeding downwards until all trace of clearness is passed. The lower edge is taken in the manner already described. The vertical measurement in the mammary line, as in deep dulness, is the one given and is known as the *superficial dulness.*

While the above is all that is usually necessary in clinical work, it is as well to state that in many cases it is quite practicable to trace the outline of the entire organ. As the lower edge of the heart lies on the liver with the diaphragm only interposing, this is by no means an easy undertaking, but when the ear and hand have become trained it can be noted that there is a difference of tone or of pitch in the dulness over the two organs which enables them to be separated.

The diagram at page 50, taken from a photograph of a patient after the heart and liver had been percussed, shows the relations of these organs, and that accuracy is quite attainable.

Enlargement of the liver occurs from congestion, waxy degeneration, fatty infiltration, malignant disease, hydatids, abscesses, and various other rarer conditions. The accompanying diagram shows the results of percussion in a case of enlarged liver from malignant disease, the stomach also being dilated. The percussion here was done on the *post-*

mortem table, and the lines were found to be absolutely correct.

Diminution in size occurs in cirrhosis, acute yellow atrophy, in prolonged venous congestion, and in diseases accompanied with starvation, as in obstructions of the œsophagus.

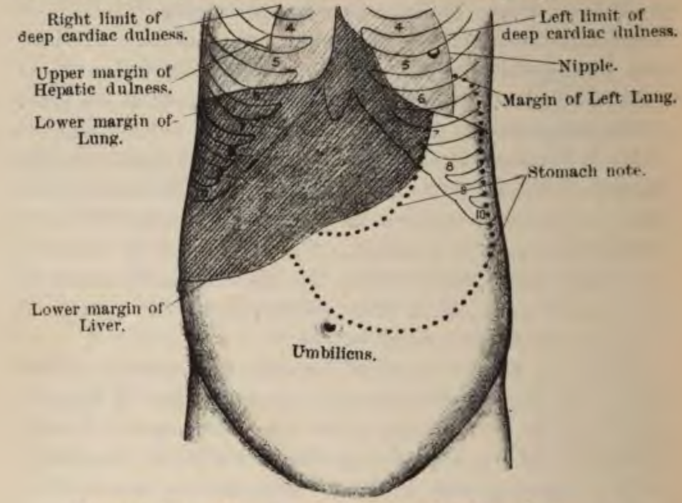

Right limit of deep cardiac dulness.
Upper margin of Hepatic dulness.
Lower margin of Lung.
Lower margin of Liver.
Left limit of deep cardiac dulness.
Nipple.
Margin of Left Lung.
Stomach note.
Umbilicus.
4
4
5
5
6
6
8
9
10

FIG. 68.—From the *post-mortem* table, showing enlargement of the liver and distention of the stomach.

THE GALL-BLADDER.

Anatomical Position.—The gall-bladder projects from the under surface of the liver opposite the ninth right costal cartilage, as shown in Frontispiece I. Its position varies somewhat: we have seen it in the parasternal and in the mammary line, or at any point between these. This, apart from individual modifications, is due to the fact that the liver is capable of lateral movement, and can be pushed to the right as far as its suspensory ligament will allow, probably by a distended stomach, as Symington has pointed out, and as we have frequently verified.

Inspection.—A distended gall-bladder may form a distinct projection on the abdominal wall, but only when the distention is great.

Palpation.—The normal gall-bladder cannot, as a rule, be felt. An enlarged one is recognised by its more or less pyriform or rounded shape, by its relation to the edge of the liver, by the fact that it moves with respiration, and by its elasticity. Its position is usually as indicated above, but we have seen it in a line with the sternum.

Causes of Distention.—Distention is due to obstruction to the outflow of the bile. The obstruction may be caused by catarrh of the duodenum, gall-stones, the *Ascaris lumbricoides*, one of the *Tæniæ*, or malignant disease of the head of the pancreas.

THE GREAT OMENTUM.

The great omentum is sometimes infiltrated with malignant growth, forming a solid cake-like structure lying on the intestines, when it is readily mistaken for an enlarged liver. The presence of a tympanitic note above it would exclude the idea of its being liver. We have also seen it drawn up, thickened, and adherent to the abdominal wall, and feeling exactly like the lower edge of the liver and mistaken for it. Further, it may be quite drawn up and form a hard thick ridge attached to the transverse colon: this occurs in some cases of diffuse malignant disease of the abdomen.

THE PANCREAS.

Anatomical Relations.—The position of the pancreas as seen from before and behind, is represented in the Frontispieces.

It crosses the vertebral column opposite the twelfth dorsal and first lumbar spines, and corresponds anteriorly with a point about three inches above the umbilicus. It is to be remembered that it is an elongated structure from one to about two inches broad, the head of which lies on the vertebral column, and a little to its right, and in the curve made by the duodenum, while its tail touches the spleen. The second part of the duodenum is thus to its right, while the third part is below it. The cœliac axis is given off above it. In close apposition to it posteriorly are the inferior vena cava, the vena portæ, and the common bile duct.

Lesions.—The most important lesion to which the organ, so far as is known, is liable is malignant disease, which affects its head chiefly. When thus affected the head forms a hard, more or less rounded swelling, which can sometimes be felt in the position already indicated. Its deep situation serves to distinguish it from a distended gall-bladder. When affected, the glands in its neighbourhood may be involved in the morbid process.

Results.—Owing to the relations of the common bile duct to it, there is obstruction to the outflow of bile, and consequent jaundice. The pancreatic duct is also occluded, and there is at times an increase of fat in the stools as a result. An increased quantity of fatty matter in the fæces is, however, much more commonly the result of hepatic disorders, or of some affection of the intestinal mucous membrane. Pressure on the vena portæ leads to ascites, but this is not constantly present, and when present it is not usually extreme.

THE SPLEEN.

Anatomical Relations.—The spleen is situated between the ninth and the eleventh ribs on the left side. Posteriorly it is in close proximity to the spinal column; anteriorly it reaches a line parallel with but a little posterior to the mid-axillary line. It measures about 5 inches in length, and 3 to 4 inches in breadth, but its size varies within considerable limits. It lies in contact with the diaphragm as the latter arches upwards. This separates it from immediate contact with the chest wall, while posteriorly the inferior edge of the lung also lies between the organ and the surface. It is in contact with the greater curvature of the stomach; and inferiorly it touches the splenic flexure of the colon. Its relations to the kidney and pancreas need not be specified, as they have no bearing on diagnosis. The organ usually presents a notch on its anterior border towards its lower end, the presence of which is of diagnostic value when the organ is enlarged and projects beyond the ribs.

Inspection.—The normal spleen gives no evidence of its presence by inspection, but when much enlarged it causes a local bulging, and may distend the greater part of the abdomen.

Palpation.—The normal spleen cannot be felt. When it is enlarged, however, it may project beyond the free edge of the ribs, and can then be felt in the left hypochondrium. It can sometimes be brought within touch of the fingers by pressing firmly with the left hand in a direction inwards and downwards over the ribs covering it. It may be so enlarged as to extend into the right side of the abdomen and downwards into the pelvis, while at the same time it may push the diaphragm upwards to such a degree that the percussion dulness reaches into the axillary region. The enlarged organ is firm, hard, and smooth to touch, and presents a well-defined notch on its anterior margin. It moves slightly during forced respiration, being pushed downwards during inspiration by the descending diaphragm, and retreating during expiration.

Percussion.—The percussion dulness of the organ measures from 3 to 3½ inches in length and 2½ inches in breadth. Percussion may be performed with the patient lying inclined to the right side, or in the sitting or standing posture. Percussion should be begun sufficiently high up in the posterior half of the axillary or infra-axillary region to elicit the pulmonary percussion sound, and continued downwards until the dull sound obtained from the organ is perceived. A mark should be made at this point. Percussion is continued in the same vertical line until a tympanitic note informs us that we have passed from the solid spleen to the intestine. This inferior limit does not quite reach the margin of the ribs if the organ be not enlarged; this fact enables us, when there is difficulty in fixing the limit of dulness inferiorly, to check our observations by means of palpation. Difficulty is experienced when the dull note of the spleen merges into and is continuous with the dull note of the colon distended with solid matter. To define the anterior limit, it is well to begin with the resonant lung sound external to the cardiac dulness at the apex, and to percuss obliquely outwards and downwards. Posteriorly the organ cannot be satisfactorily percussed beyond the scapular line, as behind this line it is separated from the chest wall by lung and diaphragm. Here also it is well to begin with the lung sound, and to percuss outwards

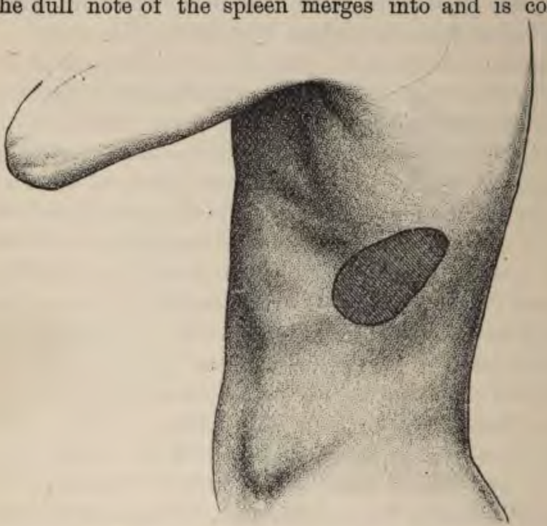


FIG. 69.—Showing the percussion dulness of the spleen. (From a photograph.)

and somewhat downwards. The annexed diagram shows the percussion of the spleen taken from a photograph.

In some acute diseases it is undesirable to move the patient on to his side. Under these circumstances we must be satisfied with the vertical dulness in the infra-axillary region, as it can be obtained with the patient lying on his back. This measurement is a sufficient index to the size of the organ, and is ample for all practical purposes.

Percussion requires to be light, for if done vigorously the tympanitic sound from the adjacent stomach or colon is brought out, and is apt to mislead the observer.

Diseases in which it is enlarged.—It is enlarged in all febrile diseases, especially ague, in pernicious anæmia, lymphadenoma, leucocythemia, general venous congestion, portal congestion, waxy disease, and rickets.

No observations have been made as to affections in which diminution in size might be regarded as of clinical importance.

THE KIDNEYS.

Anatomical Relations.—The position of the kidneys is represented in Frontispiece II. They are situated in the loins, and lie against the posterior wall of the abdomen at the level of the last dorsal and two or three upper lumbar vertebræ. At their concave (inner) surface they are in close relation to the transverse processes of the vertebræ. The upper border of the right kidney touches the under surface of the liver, while the left one touches the spleen. They are about four inches long and about two and a half broad. They are outside the peritoneum.

Palpation.—The kidneys are palpated by pressing one hand firmly into the loin covering them, and the other deeply from the front, so as to try to feel the organ between the two hands. The normal kidney, even in very thin persons, can only be indistinctly felt, but even when it cannot be satisfactorily felt, the production of pain by palpation is often of great diagnostic value in renal calculus, and in the early stages of strumous disease before the organ is much enlarged. When they are much enlarged they can be distinctly felt. If they are the seat of hydronephrosis, pyelonephrosis, or if their substance is destroyed by large abscesses, as in strumous nephritis, not only can their enlargement be felt, but distinct fluctuation can be obtained with the hands in the position as directed above. They are also readily felt when enlarged from malignant disease.

Percussion.—Percussion of the normal kidneys, as might be inferred from the thickness of the muscular structures behind them, and their close relation to the vertebral column and some of the solid organs in the abdomen, is unsatisfactory, and is not relied upon in clinical work. The lower limit of the organs can, however, be made out posteriorly owing to their relation to the colon, the commencement of its tympanitic sound marking the inferior extremity of the kidney. In the same way the convex surface of the organs may be made out. Percussion requires to be strong, and is perhaps best performed with a plessor and pleximeter. When the organs are enlarged percussion is more feasible; when the enlargement is great the dull percussion sound can then be readily elicited even in front, but even then greater reliance is placed on palpation.

Floating or loose kidney is a condition which is occasionally met with. It occurs most commonly on the right side. It results from the looseness of the renal connections permitting the organ to leave its normal position, and to come forwards until it can be felt lying under the edge of the liver; or it can be pushed out of its normal position by one hand in the loin, while it is felt through the anterior abdominal wall by the other. It can sometimes be pushed across the middle line. That the organ is the kidney is recognised during life by its mobility, position, shape, and sometimes by the sensation of nausea caused by grasping it firmly. The flank may be less dull on percussion than the other one. The percussion sound over the kidney itself when it is near the anterior abdominal wall is of course dull. Attacks of peritonitis sometimes occur round it, and cause pain and tenderness on palpation.

Perinephric Abscess.—This subject requires slight reference under this section. Apart from the constitutional symptoms, there are tenderness on pressure, fulness, and, in marked cases, even fluctuation when the renal region is palpated. The constitutional symptoms and the acuteness of the onset usually distinguish it from suppurative inflammations of the kidney substance. The area of dulness should be increased, but even here this means of diagnosis is unreliable.

THE SUPRARENAL CAPSULES.

These organs are situated on the antero-superior surface of the kidneys. They may be the seat of malignant or strumous disease, and if their enlargement is considerable they may, in thin persons, be felt by careful palpation.

ABDOMINAL ANEURISM.

The abdominal aneurisms which come under the care of the physician are those of the abdominal aorta or of the cœliac axis. The most commonly affected part of the aorta is just below the diaphragm, and in this situation it involves the cœliac axis. Aneurism is recognised by the presence of abnormal pulsation; by the presence of a tumour, the pulsation of which is distensile; sometimes by the presence of a thrill; and by the presence of a systolic murmur over it. The time and the fulness of the pulse in the femoral arteries, as compared with the arteries in the upper limb, may be of assistance; in aneurism the pulse in the former may be delayed slightly, and be less full. In addition, aneurism may produce great pain by pressure on nerves and on the vertebral column, the bones of which may become eroded. When this last occurs, there is tenderness on pressure over the spine, and there may even be curvature. If the aneurism be large, it may exercise pressure on other important structures, such as the large veins in the abdomen.

In the epigastrium pulsation is present in many cases in which there is no aneurism. In enlargement of the right ventricle and in downward displacement of the heart it is usual to have pulsation here. In thin persons it is also by no means uncommon to have very marked pulsation of the upper part of the abdominal aorta. In these no tumour can be felt, and there is an absence of other confirmatory evidence of aneurism. When a tumour of the liver, or in fact any abdominal tumour, is in contact with the aorta, the pulsations of the vessel are communicated to it and to the surface, and the condition is very readily mistaken for aneurism. It is specially to be remembered that the pulsation of an aneurism is distensile, and that, if there are not other evidences of the pulsation being aneurismal, the diagnosis ought to be made with great caution.

BLADDER.

In many diseases it is important to pay particular attention to the state of the bladder. More particularly is it necessary to bear in mind that retention of urine occurs in various injuries and diseases of the spine, in the delirious stage of the acute fevers, and in all more or less comatose states. The distention of the viscus is recognised by dulness on percussion above the pubes; in extreme distention it may produce a very marked bulging of the hypogastrium, and may reach even as high as the umbilicus. Care must also be taken that the constant dribbling away of the urine does not lead one to think that the viscus is emptying itself, when only the *overflow* is passing away.

CONTENTS OF THE ALIMENTARY CANAL.

Under the above head fall to be considered the examination of matters voided by the stomach and rectum, and the investigation more especially of the contents of the former.

Vomiting—Causes.—Vomiting may be due to very diverse causes, the investigation and differentiation of which are often surrounded with difficulty and uncertainty. It may be due—(*a.*) To local disorders of the stomach: (*b.*) To affections of other parts of the digestive system, as in intestinal obstruction, biliary colic, and peritonitis: (*c.*) To affections of the central nervous system, as in uræmic poisoning, meningitis, tumour, and hæmorrhage at its onset, while in rare cases it is periodic and epileptoid in type: (*d.*) To organs not concerned in digestion, as the uterus, pregnancy being often associated with very troublesome vomiting, and the kidney, renal colic being commonly accompanied with vomiting; in these the action is a reflex one: (*e.*) To the poisons of some of the acute fevers, and poisons introduced into the stomach, given as emetics, taken accidentally, or administered with criminal intention.

Time and manner.—The time and manner of vomiting provide important indications as to its cause. It may occur almost immediately after taking food, and may or may not be accompanied or preceded by pain, when it indicates that some local irritative condition of the stomach exists. It may be delayed until one, two, or more hours after a meal, when it is usually preceded by a sense of distention, perhaps pain, acidity, flatulence, or heartburn; in such cases the digestive process in the stomach has been imperfectly performed, or the digested material has not passed out of the stomach. In the first the local cause may be catarrh, ulceration, or malignant disease of the body of the viscus; in the latter it is due to obstruction at the pylorus from malignant thickening or from the cicatricial contraction following upon the repair of a simple ulcer. Relaxation with dilatation from simple atony also leads to retention of the gastric contents. In all these vomiting is preceded by some premonitory symptom referable to the stomach, as pain, nausea, or discomfort; when due to other than stomachic causes, there is usually no symptom referable directly to it. In biliary and renal colic the vomiting accompanies the pain, and is a reflex of it. When it is neurosal in origin it is usually unaccompanied by nausea or pain. In this form, and in that due to colic, it, as a rule, occurs independently of taking food; but this is by no means invariably the case, for it can often be noted that, even when purely neurosal, as in meningitis, the vomiting may occur immediately after taking food, or even water, into the stomach, although it may reveal its true nature by occurring also at times when food has not been taken.

Characters of the vomited matters.—These vary according to the length of time the food has lain in the stomach, the characters of the food partaken of, the secondary changes which may have supervened, and the nature of the stomach lesion.

They may consist of clear sour fluid, or contain a variable amount of stringy mucus.

They may consist of the food which had been taken, the different articles being in different stages of solution and digestion, when they emit a heavy sour odour.

The green or yellow colour which vomited matters frequently present is due to their admixture with bile.

When blood is vomited the condition is known as hæmatemesis. It may be almost pure and fluid, when the hæmorrhage has been severe, but as a rule in such cases it is clotted. This occurs when a large vessel is eroded, as in gastric ulcer, and in extreme congestion, as from cirrhosis of the liver. On the other hand, it may be mixed equally with the fluid or semifluid contents of the stomach, giving them a uniform brown or blackish colour when it is usually described as coffee-ground vomit. This may be its character in gastric ulcer, in congestion, or in acute inflammation.

It is necessary to remember that blood vomited from the stomach may not be derived from its vessels, but have been swallowed.

In some cases, if the gastric contents are long retained, as in dilatation, they undergo a second fermentation. When this has occurred and vomiting ensues, the vomited matters have a thick covering of froth resembling barm, with a smell not unlike that of beer. The fermentation is due to the presence of the *Sarcinæ ventriculi*, which is found abundantly on microscopic examination.

In cases of intestinal obstruction there is usually very severe vomiting, the vomited matters after a time consisting of the fæcal contents of the small intestine, when it is known as stercoraceous vomit.

Investigation of the contents of the Stomach.—The stomach may be emptied of its contents by means of the flexible œsophageal catheter, joined by a piece of glass tubing to an indiarubber tube, into the end of which a glass funnel may be inserted if necessary. If the stomach is at all distended the contents flow out at once on the introduction of the catheter, but if the viscus contains but little food, it may be necessary to apply a certain amount of pressure to the abdomen in order to expel the substances in it, or even to wash the stomach out. This is done by raising the tube and funnel to a level above that of the patient's head and pouring water into the funnel. The water descends by gravitation into the tube and distends the stomach. After a quart has in this way entered the stomach, the funnel and tube, while still full of water, are to be lowered over a vessel ready to receive the gastric contents, when the apparatus acts as a syphon and drains the stomach. This is to be repeated until the water comes away from the stomach perfectly clear.

Care must be taken only to employ this method in suitable cases, and to avoid using it in any cases of structural change in the walls of the stomach. The catheter should never be introduced without the tube attached, as cases are on record in which the patient has swallowed the catheter entirely.

To ascertain the duration of gastric digestion, the stomach is emptied at the time when the process should be at an end. For the performance of this examination it is necessary to provide the patient with a standard meal, such as a plate of clear soup, a chop or beef-steak, and a dinner roll, and to see that no food is taken in the interval before examining the gastric contents. Seven hours afterwards the stomach is to be emptied or washed out. If perfectly empty the process may be repeated the following day, two hours earlier than on the first attempt, and this may be done again until the exact duration of gastric digestion is ascertained.

If the stomach at the end of seven hours contains nothing more than a few shreds or flakes, the digestion may be held to be nearly normal. This shows that there is no interference with the exit of food, and that the gastric secretion is sufficiently active. If there be any considerable remains of food in the stomach, it is clear that the digestion is retarded by inactivity of the secretion, weakness of the peristalsis, obstruction of the pylorus, or paralysis of the muscular wall.

The condition of the gastric contents during digestion may be ascertained by introducing the catheter during the digestive process and drawing off a small quantity. This is first examined as it stands to observe if any bile, blood, or other extraneous substance is present. It is then filtered.

The *solids* on the filter are examined both by the naked eye and by the microscope, to ascertain the degree of maceration and trituration, as well as to detect the presence of parasitic growths, as already mentioned.

The *filtrate* must then be examined. The *reaction* must be determined by means of litmus paper, but as the acidity may be caused by organic acids as well as free hydrochloric acid, further tests must be employed.

For *free hydrochloric acid* several tests are now in use, such as methyl-violet, phloro-glucin-vanillin, tropœolin, and other reagents. The two first named are the most satisfactory.

An aqueous solution of methyl-violet of a strength giving a medium shade of colour is used. Two test-tubes are half-filled with the reagent, and to one of them is added about half its bulk of the filtrate. If the gastric secretion is normal, the solution of methyl-violet should assume a distinctly blue colour. If this blue colour is not clearly struck, the secretion is wanting in free hydrochloric acid. It is to be remembered that albumins and peptones hinder the reaction, and that common salt occasionally gives a reaction similar to that of hydrochloric acid. The organic acids give no reaction unless in a state of concentration unknown in the stomach.

The phloro-glucin-vanillin test is employed as follows:—The solution is made by dissolving two parts of phloro-glucin and one part of vanillin in thirty parts of rectified spirit. A few drops of this solution are mixed with an equal bulk of the filtrate in a porcelain plate, and the mixture is evaporated over a water bath. If the filtrate contains a normal amount of free hydrochloric acid, a bright red colour is left as a deposit.

Lactic acid may be detected by the ferric chloride and carbolic acid test. A mixture containing a few drops of weak ferric chloride solution added to about 100 cubic centimetres of a one or two per cent. solution of carbolic acid, gives a bluish-grey colour. On adding some of the filtrate to this, if lactic acid is present, a yellowish colour is produced. If hydrochloric acid alone is present, the mixture becomes colourless.

Further tests may be employed, but these must be sought for in treatises devoted to the digestive system.

The tests for *pepsin* and *peptones* are far from satisfactory, and cannot be dealt with in this place.

The absence of the reaction with methyl-violet or phloro-glucin-vanillin points to some serious alteration in the functions of the stomach. An increased reaction points to hyperacidity, but the bearings of such a condition are still under dispute.

An excess of lactic acid, as shown by great rapidity of reaction and brightness of colour struck, points in most cases to retarded digestion and dilatation of the stomach.

The Alvine Discharges.—It is always necessary to make inquiries regarding these, and not infrequently it is necessary to examine them.

The discharge of blood from the bowel is known as melæna: it may be almost pure, or mixed with the fæcal discharges, making them more or less dark in colour. It may be due to anal fissure, piles, simple or malignant ulceration, or the ulceration of typhoid fever or dysentery, to severe congestion, as in cirrhotic liver, to acute inflammation of any part of the intestinal tract, or to intussusception.

Mucus may be passed in excess, and be accompanied with extreme straining; this occurs in dysenteric diarrhœa, when it may also be mixed with a variable amount of blood.

Parasites of various kinds may be passed with the fæces or alone; the commonest are the thread-worm (*Oxyuris vermicularis*), which inhabits the rectum; the round-worm (*Ascaris lumbricoides*), which inhabits the small intestine usually; and the tape-worms (*Tænia solium* being the commonest), which extend through small and large intestines.

When drugs are administered for the removal of tapeworms it is desirable to examine the intestinal discharges for the head of the parasite, as the cure is not completed until it has been expelled. This is done by thoroughly mixing the discharges with water and straining them through muslin; the head is to be carefully looked for in the retained matters on the muslin, and it requires care, as it is no larger than the head of a small pin; it often, however, has several segments attached to it, when its discovery is easy.

The fæcal discharges from the bowel are, however, those which it is oftenest necessary to examine. For the perfectly healthful state of the body it is necessary that there should be thorough evacuation of the fæcal contents of the intestines every twenty-four hours. The characters of these discharges, as regards consistence and colour, vary greatly within what is presumably normal limits. In constipation the amount passed is small, and of firmer consistence and lumpy; while in diarrhœa the amount is increased, the evacuations being fluid or semi-fluid. In typhoid fever the stools may closely resemble pea-soup, and are known by that name. In cholera the evacuations are watery, with flakes of mucus floating in them, and from these naked-eye characters are known as rice-water stools. In jaundice, from obstruction to the outflow of bile, the stools are whitish or clay coloured.

The evacuations may be examined microscopically for micro-organisms, or plate cultures may be made to separate the various varieties.

Chemical examination may be undertaken for the purpose of determining the quantity of fat and other ingredients in them.

CHAPTER VIII.

THE URINARY SYSTEM.

THE results obtained by examination of the urine serve a two-fold purpose. In their wider significance they furnish evidence in regard to the processes of tissue change in the system at large, while in their narrower application they provide the most important, and frequently the only, guide to the condition of the urinary organs. A thorough acquaintance with the methods of ascertaining the physical and chemical characters of the urine is therefore of the highest value. In the following pages attention will be called to the various modes of procedure to be adopted in its clinical investigation.

Method of Examination.—In making a systematic examination of the urine, some plan should be adopted, such as the following:—

Determine the daily quantity.
Observe the appearances as to colour, clearness, and deposits.
Ascertain if there is any peculiar smell.
Take the specific gravity.
Note the reaction.
Proceed to the chemical examination, especially with regard to the amount of urea, and the presence or absence of albumin and glucose.
Examine both the urine and its deposits by means of the microscope.

For the complete examination of the urine the following apparatus will be found necessary :—

Vessels for collecting urine.
Tall urine-glasses.
Conical glasses.
Measures graduated in cubic centimetres.
A graduated burette.
Test-tubes of various sizes.
Beakers.
Watch-glasses.
Glass rods.
Pipettes.
Porcelain plates.
Spirit lamp.
Test papers.
Funnels and filtering paper.
Urinometer.
An apparatus for estimating urea.

The following solutions are also required:—

The solution of caustic soda for urea testing.
Bromine.
Pure nitric acid.
Fuming nitric acid.
Pure hydrochloric acid.
Pure sulphuric acid.
Pure acetic acid.
Liquor potassæ.
Liquor ammoniæ.
Fehling's or Pavy's solution.
Solution of silver nitrate 1 to 8.
Other special solutions, required for volumetric analysis of different substances, will be mentioned under their respective heads.

Method of obtaining Samples.—In order to have a true estimate of the state of the urinary secretion, it is necessary to examine specimens taken from the total quantity passed during a period of twenty-four hours. The patient should be directed to empty the bladder at a certain hour, and to reject the specimen then passed; after which the urine is to be collected until the same hour of the following day, when the last sample is to be added to the vessel employed for the purpose. The total quantity of the mixed urine is to be measured, and specimens are to be laid aside for careful examination. It is often desirable to have the urine passed during the night and the day separately examined, and it may in certain cases be advisable to determine the characters of that secreted before and after food, for which purpose separate specimens must be collected. It may be necessary to ascertain whether deposits have their origin in the upper or lower urinary passages; and, with this end in view, the patient should be ordered to pass the first two ounces of urine into one vessel, and the rest into another. By this means it is easy to distinguish the products of the urethra from those of higher origin, as the portion first passed will contain almost all the substances which have accumulated in the urethra. It may even be advisable to obtain specimens of urine free from any substances derived from the walls of the bladder. This may be done by drawing off the urine by means of a soft catheter while the patient stands, after which the bladder is to be washed out by several injections of small quantities of warm water, and the urine, which subsequently trickles drop by drop from the catheter, is to be collected. Obtained in this way it is practically free from any of the products of the bladder.

Physical Characters of Healthy Urine.—The quantity of urine passed by an adult in twenty-four hours varies with the amount of fluid taken and the amount of water excreted by other channels, and in each individual case these two factors must be considered. The average amount is about 1500 cubic centimetres, or about 50 ounces. When newly passed, the urine is a clear fluid having a yellowish colour, a peculiar aromatic smell, an acid reaction, and a specific gravity depending on the amount of solids in solution, and therefore very variable, being frequently, in perfectly normal conditions, temporarily as low as 1005 and as high as 1030. The average specific gravity is about 1020. On standing in a glass vessel for eight or ten hours a faint cloud may be seen gradually to form in the lower portion of the urine. This is composed for the most part of *mucus.* This delicate cloud remains unchanged when acted on by heat, alkalies, and strong acids, but acetic acid sometimes increases it by coagulation of the mucin.

If the urine is alkaline it is somewhat cloudy when passed from the deposition of *earthy phosphates.* These at once begin to fall down and form a hazy stratum, which gradually subsides to the bottom of the vessel. The true nature of this appearance may be detected by the addition of a few drops of an acid which causes the cloud to disappear.

It may, soon after being passed, become muddy from the precipitation of *mixed urates,* which are not so easily soluble in cold as warm water. The turbidity in this case passes gradually away from the falling of the urates to the bottom of the vessel. Urates are at once recognised by the fact that on heating the turbidity disappears.

Certain changes in appearance which healthy urine undergoes on exposure to the air must be described. When a specimen of healthy acid urine is allowed to stand for some days, it develops the hazy or nebulous cloud previously referred to. It afterwards assumes throughout a darker colour, and usually throws down a deposit of a ruddy tint, which is found on microscopic examination to consist of uric acid crystals. At this stage the reaction is much more acid, lactic acid is formed in the urine, and the changes just described constitute the *acid fermentation* of the urine.

These appearances are followed by another series of changes termed the *alkaline fermentation,* which may set in, however, at once after the passing of the urine, or even before it has left the bladder. Apart from the decomposition caused by the presence of organisms in diseased conditions of the urinary organs, the alkaline fermentation of the urine sets in more quickly when the external temperature is high, when the urine has been placed in an unclean vessel, and when mucus, pus, or blood is present. The dark coloured urine becomes paler, the uric acid deposit is replaced by a dirty grey sediment, which often, by transmitted light, may be seen to contain shining crystals, and on the surface floats an iridescent pellicle. In the sediment and pellicle the microscope reveals numerous crystals of calcium phosphate, ammonio-magnesium phosphate, calcium oxalate, and ammonium urate, while the urine swarms with bacteria. A strong smell of ammoniacal urine is given off, and the urine gives red litmus paper a blue colour; indeed, the red litmus may be changed in colour merely by holding it while damp for a short time above the fluid. The blue colour gradually fades when the litmus paper dries, showing that the alkali which has caused the reaction is volatile, and the cause of the change is that during the process of fermentation the urea takes up water, and becomes converted into ammonium carbonate—

$$CH_4N_2O + 2H_2O = 2(NH_4)CO_3.$$

Chemical Characters of Healthy Urine.—The amount of solids contained in the urine of twenty-four hours depends largely upon the amount of food taken. On an average diet it varies between 60 and 70 grammes, or 900 and 1050 grains. Rather more than one-half of this consists of *organic substances.* These are numerous, but those which alone possess any importance in clinical medicine are urea, uric acid, hippuric acid, kreatinin, xanthin, lactic acid, oxalic acid, urobilin, and organic sulphur-containing bodies, such as indican. Urea forms by far the largest proportion of the organic constituents, its amount being from 28 to 40 grammes, or from 420 to 600 grains in twenty-four hours. The other organic substances taken together scarcely amount to 3 grammes or 45 grains in the same period.

The *inorganic constituents* form, roughly speaking, about half of the total solids. The most important of these are chlorides and sulphates, phosphates of the alkalies and alkaline earths, with smaller quantities of iron, free ammonia, and carbonic acid. Sodium chloride forms a large proportion of the inorganic constituents of the urine, the daily quantity passed varying from 10 to 15 grammes, or from 150 to 225 grains; that is, about one-half of the inorganic, or about one-fourth of the total solids.

Variations in the Physical Characters of the Urine.—Alterations in the physical properties of the urine depend on changes in its chemical composition, and serve to point out the directions in which such chemical modifications have taken place. It will be necessary to describe variations in the quantity, colour, smell, reaction, and density of the urine.

1. *Variations in the Quantity of the Urine.*—The amount of fluid taken and the amount excreted by other channels is the great source of variations in the quantity. The entrance of fluid into the system is followed by an increase, and any great loss by the skin or bowels by a diminution in the amount of urine. Changes in the structure of the kidney give rise to alterations in the amount of its secretion. The urine may be greatly increased in quantity in waxy disease, and entirely suppressed in acute inflammation. From the conditions of its secretion it is clear that any modification of the circulation must be followed by a change in the amount of the urine. Increase of the blood pressure, as in cirrhosis of the kidney, causes an increase; while diminished blood pressure, as in cardiac diseases, lessens the amount. Nervous influences acting on the circulation cause variations in the flow of urine. Amongst these may be mentioned hysterical polyuria and oliguria, the increase which is sometimes found as a symptom of cerebral lesions, and the conditions known as diabetes mellitus and diabetes insipidus. Suppression of urine may be caused by some obstruction in the urinary channels, such as a calculus in the pelvis or

ureter, or it may be caused by organic disease of the kidney, by certain acute febrile disorders, or by shock and collapse.

2. *Variations in the Colour of the Urine.*—Changes in colour may be caused by alterations in the relative proportions of the colouring matters and the water of the urine, or by the presence of abnormal substances.

The principal colouring matter of the urine in health is *urobilin*, derived from the bilirubin of the bile, and the depth of colour varies with the proportion of this substance to the water. When the urine is increased in quantity, as in winter from lessened perspiration, in free drinking (*urina potus*), in simple polyuria, in diabetes insipidus, in diabetes mellitus, in renal cirrhosis and waxy degeneration, its colour is pale. When, on the contrary, there is a diminution in the amount of urine passed, as in summer from free perspiration, in febrile conditions, and in the scanty urine of certain renal and circulatory diseases, the colour is deep. The morning urine (*urina sanguinis*) is always more highly coloured than that passed during the day. There are some other pigments, related to urobilin, and probably arising from the same source, but they do not at present concern the physician in any practical sense. For a description of them the reader may be referred to an excellent paper by MacMunn.[1]

Another important chromogen is *indican*, which owes its origin to indol, a product of pancreatic digestion. The proportion of indican is always greater when the urine is lessened in quantity, when the food is highly nitrogenous, and in many diseases, especially in intestinal obstruction. Some coloured urines, and more particularly those of a blue or violet colour (*glaukuria*) are believed to be due to the oxidation of indican. The methods of determining the presence of urobilin and indican will be described at a later stage.

Reddish urines owe their colour to the presence of *blood*, which may have its origin in various parts of the urinary organs, and may accordingly present different appearances. Blood arising from the walls of the urethra is not mixed with the urine, but appears as a small clot at the beginning of the act of micturition. When the blood has its origin in the bladder the first part of the urine passed is usually unaltered in appearance, but that discharged towards the end of micturition is deeply coloured, and commonly contains coloured clots of various shapes. When springing from a higher source the blood is diffused throughout the urine and blood-clots are rarely present. There are, however, cases in which clots, having their origin in the ureter or in the pelvis of the kidney, may be found, but in such instances the coagulated blood is decolorised and has an elongated shape from being moulded in these passages. When blood corpuscles are seen, under the microscope, adhering to renal tube-casts, as will be afterwards described, there can be no question as to the source of the blood. It must not be forgotten that the urine may contain blood which has flowed from the vagina, and the fact must not be overlooked that blood may be added to urine in order to deceive the medical attendant. In such cases it is not difficult by the exercise of a little care to trace the blood to its origin.

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[1] *British Medical Journal*, 1888, vol. ii., p. 117.

When clots are present in the urine they should always be floated upon water to facilitate the recognition of their source. In this way it is easy to distinguish their outline, and from this to determine the seat of their origin.

When blood has its source in the kidney it is intimately mixed with the urine. In one class of cases the microscope reveals the blood corpuscles, which may or may not be altered in outline, but never form rouleaux. Such cases are known as *hæmaturia.* In another class only the granular and pigmented débris of the broken-down corpuscle is seen. This condition is termed *hæmoglobinuria.*

In hæmaturia the colour varies greatly, and the urine is never clear. When blood is only present in small quantity the urine is smoky in appearance or dingy in colour, and there is a perfect gradation between this tint and deep red or brown shades. As a rule, with an equal quantity of blood, acid assumes a deeper tint than alkaline urine. In hæmaturia albumin is, as might be expected, always present in the urine; epithelium, mucus, or pus may often be found; and tube-casts are to be seen in certain cases. On standing the urine deposits a reddish or brownish sediment, which should be carefully examined. In endemic hæmaturia, as will be seen in a subsequent part of the work, the ova of the *Bilharzia hæmatobia* are present in the urinary deposit.

The urine in hæmoglobinuria assumes a brownish tint, often said to resemble the colour of port, and is as a rule quite clear when passed. It always contains albumin, but not in proportions so great as in hæmaturia, and throws down on standing a ruddy deposit consisting of epithelium and tube-casts, with the occasional presence of hæmatin crystals. The hæmoglobin is held by Hoppe-Seyler to be in the form of methæmoglobin.

When the *colouring matters of the bile* are present in the urine they impart to it different shades of colour varying from *yellowish brown* to *brownish* or *greenish black*. If white blotting-paper or white linen is dipped in such icteric urines a bright yellow colour is imparted to it, and if they are violently shaken, the froth formed on the surface, which usually remains there for a considerable time afterwards, assumes a bright yellowish or greenish colour. The bile pigment may or may not be accompanied by bile acids, and the chemical tests for these substances will be described below.

A *milky appearance* is given to the urine by the presence of finely divided *fatty matters* in the condition known as *chyluria*, and this appearance has frequently a ruddy tinge from the presence of blood. In this condition there is usually to be found on standing a layer of finely divided fatty matter upon the surface of the fluid. Even although imperceptible to the unaided eye, blood is always to be detected, and as a necessary consequence albumin is present.

Sometimes the fatty matter may be present in such a form as to present the appearance of large drops of oil diffused through and tending to accumulate on the surface of the urine instead of being in smaller globules.

A deep reddish-brown colour is given to the urine by the presence of *pyro-catechin*, which is more observable when it has been allowed to stand exposed for some time to the air. It appears still to be an open question whether pyro-catechin is the same substance as *alkapton*, a yellowish body with aromatic properties, which gives the urine a deep brownish tint on standing.

A very *dark*, even *black*, colour may be given to the urine by the presence of *melanin* in cases of chronic ague and melanotic cancer. On standing, the urine, which is at first clear, throws down a deposit of dark granules, the exact nature of which is still shrouded in obscurity.

It must not be forgotten that the use of certain substances may tinge the urine. Senna and rhubarb, by means of their chrysophan, give a deep yellow tint to acid and a bright carmine to alkaline urines. Santonin and picric acid produce a brilliant yellow, while juniper gives a greenish-yellow colour. Logwood with alkaline urine gives a blue or violet colour, and carbolic acid in large doses is the cause of a greenish-black colour. Many of the modern aniline preparations also colour the urine.

3. *Variations in the Odour of the Urine.* — Little is accurately known in regard to the smell of the urine. Under ordinary circumstances it has an odour which may be termed aromatic, and which varies in its intensity very considerably. When the alkaline fermentation begins, there is an ammoniacal smell, and in cases in which decomposition of the urine takes place before it is passed the odour may be intensely putrid, or even sulphurous, from the presence of sulphuretted hydrogen.

The urine frequently assumes the smell of substances which have been taken internally. Radish, onions, and other vegetables give the urine a characteristic odour, while asafœtida, valerian, and many other aromatic drugs act in a similar manner. It must not be forgotten that turpentine and its congeners produce a smell not altogether unlike that of violets in the urine.

4. *Variations in the reaction of the Urine.*—In health the reaction of the urine is almost invariably acid, probably from the acid phosphate of sodium which it contains, although no doubt the presence of carbonic acid, from the conversion of organic acids, increases it. The reaction varies, however, at different periods of the day, and becomes less acid or even alkaline after a full meal. It is a well-known fact that in carnivora the urine is highly acid, while it is alkaline in herbivora.

The acidity of the urine may be shown by its action on blue litmus paper, and may be estimated quantitatively by means of a standard solution of soda.

The acidity of the urine may be increased relatively or absolutely. Whenever the quantity of the urine is diminished, as in hot weather from free perspiration, it becomes more highly acid. On the other hand, the acidity may be absolutely increased in disorders of the functions connected with food digestion and tissue change. Similarly the amount of acid present may undergo diminution either in an absolute or relative sense, from reversed conditions.

Alkalinity of the urine may be caused by the presence of a fixed or a volatile alkali, and may be shown by its action on red litmus and on turmeric paper. Permanently alkaline urine, so called from the fact that the blue stain given by it to red litmus paper does not disappear on drying, is due to the presence of carbonates of potassium and sodium in excess. The causes of the excessive elimination of these salts are numerous, and it need only be stated here that besides being the result of the internal use of alkalies and their salts, taken either in the food or as drugs, they have their origin primarily or secondarily in disturbances of the processes of digestion and assimilation.

Temporary alkalinity of the urine is produced by the presence of carbonate of ammonium, and the blue colour which it gives to red litmus paper fades on drying. Decomposition of urea while the urine is in the bladder, or after it has been passed, is the sole cause giving rise to this volatile alkalinity of the urine. It has its origin in the presence of an organism (*Micrococcus ureæ*), and cannot take place unless there be a previously unhealthy state of the urinary tract. The ammoniacal fermentation of the urine always gives rise to the presence of deposits of calcium phosphate and ammonio-magnesium phosphate. It is accompanied by a film, usually somewhat iridescent in appearance upon the surface, which consists mainly of these two earthy phosphates.

5. *Variations in the Specific Gravity of the Urine.*—The specific gravity of the urine is ascertained by means of a modification of the hydrometer specially adapted for the purpose, and termed, therefore, a *urinometer*. The scale which it contains should begin with the specific gravity of water, taken as 1000, as zero, and should register as far as 1050. The only other point which need be referred to in connection with it, is that it should be carefully tested in distilled water before it is employed to examine urine, and that urine should be tested by it at a temperature of 15·5° C. or 60° F., as for every 3° C. or 5·4° F. above that temperature the specific gravity falls one degree.

The specific gravity depends upon the relative amount of the fluid and solids of the urine, and in health usually varies between 1015 and 1025, the normal specific gravity of the healthy urine of twenty-four hours being 1020.

The proportion of solids in the urine, as we have already seen, is on an average 60 grammes in 1500 cubic centimetres, or 1 in 25,—that is, four per cent. As the normal specific gravity of the urine is 1020, we find a simple method of estimating the approximate quantity of solids by multiplying the two last figures of the specific gravity by two, in order to obtain the bulk of solids in 1000 cubic centimetres. Having done this, it is easy to calculate the amount of solids in any given volume of urine. Normal urine, with specific gravity of 1020, gives 40 grammes as the amount of solids in 1000 cubic centimetres, and therefore 60 grammes in 1500 cubic centimetres, as obtained by the following formula :—

$$1000 : 1500 :: 40 : 60.$$

This calculation is commonly known as the *co-efficient of Trapp ;* in urine with a specific gravity exceeding 1025 more accurate results are obtained by using the *co-efficient of Haeser,—i.e.*, by multiplying the last two figures by 2·33.

Marked and persistent deviations from the average specific gravity point to certain diseases as being probably present. The specific gravity may be much increased while the amount of urine undergoes little or no change in certain disorders of metabolism resulting in the production of an excess of urea, uric acid, and urates. Increased specific gravity, with lessening of the quantity of urine, is found in acute febrile disorders, and in acute renal disease with the presence of albumen. Greatly increased specific gravity, with an increased flow of urine, points to the probability that diabetes mellitus is present. Diminution of the specific gravity, without much alteration in the bulk of the urine, is found in the early stage of renal cirrhosis, which is probably the same condition as that which has been described under the name of renal inadequacy. In it the amount of urea is below the average. Diminished specific gravity with lessened amount of urine may almost be said to be unknown, while with increased quantity of urine it points to a later stage of cirrhosis of the kidney than that just referred to, after vascular and cardiac hypertrophy have advanced ; to waxy disease ; to diabetes insipidus, in which the total solids are not absolutely diminished ; or to hydruria from nervous or other causes, in which the solids are absolutely lessened. The urinometer in these last two disorders affords great aid in diagnosis. The specific gravity may remain normal while the amount of urine is greatly increased in the condition known as polyuria. The causes of this condition are to be sought for in greater rapidity of tissue change, with consequent increase of waste and urinary solids.

Variations in the Chemical Characters of the Urine.—The urine may depart from the healthy condition in two ways,—on the one hand, by alteration in the quantity of the normal constituents; and, on the other, by the presence of abnormal substances. In the following pages the usual methods of determining the presence and quantity of the urinary constituents will be given in the order above referred to.

The normal constituents of the urine, to which, for practical purposes, attention must be directed, are, among organic substances, urea, uric acid, hippuric acid, kreatinin, xanthin, lactic acid, oxalic acid, and the colouring matters, urobilin and indican; among inorganic substances, chlorides, sulphates, and phosphates.

Urea (CH_4N_2O).—The presence of urea can always be *demonstrated* by evaporating the urine, if of ordinary specific gravity, to about half its bulk, and adding an equal quantity of nitric acid. On cooling the mixture, shining rhombic crystals of nitrate of urea are seen throughout the fluid. This reaction, as will be seen below, is apt to mislead the observer in testing for albumin. If the urine is of high specific gravity, it is often unnecessary to concentrate it by evaporation previous to the addition of nitric acid.

In order to obtain urea in a pure state, it is necessary to precipitate the sulphates and phosphates by the addition of a saturated solution of barium nitrate and barium hydrate, and remove them by filtration. The filtered urine is then evaporated until it becomes syrupy, when an equal bulk of strong nitric acid is added to it. The crystals of urea nitrate, which are formed by this means, are decomposed by the addition of a solution of carbonate of barium and subsequent boiling, after which the fluid must again be concentrated. It is then mixed with boiling alcohol and filtered through animal charcoal. On evaporation pure urea is deposited.

It assumes, as in Fig. 70, the form of prismatic crystals, often branching into fan-like masses, and is very soluble in water, the solution being neutral. It forms salts with acids which are by no means so soluble as urea.

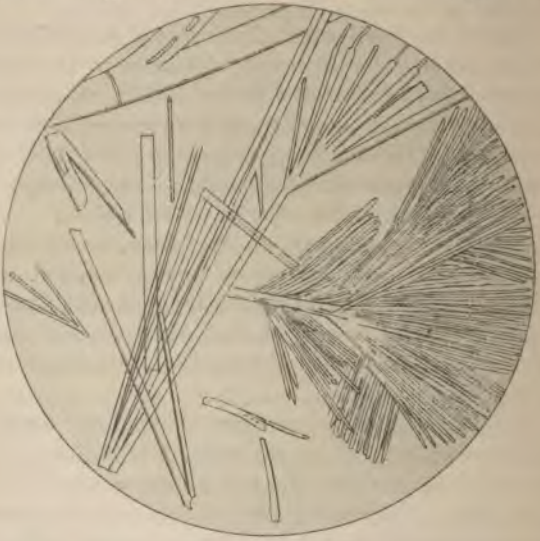

FIG. 70.—Urea.

The amount of urea may be ascertained by precipitating it as an insoluble compound of mercury by the method of Liebig, or by decomposing it by means of sodium hypobromite or hypochlorite, and estimating the quantity from the volume of nitrogen given off, as was first proposed by Davy.

Quantitative analysis by precipitation is based on the fact that certain salts of mercury form a fixed compound with urea which is insoluble; and the method employed is to use a solution of nitrate of mercury of definite strength, corresponding to a certain amount of urea, the completion of the precipitation being known when the mixed solutions give with sodium carbonate a yellow colour from the formation of oxide of mercury. It is necessary, before employing this method, to remove all phosphates, sulphates, and chlorides from the urine.

For the performance of this test the following solutions are necessary:—

A saturated solution of barium nitrate and barium hydrate, in the proportion of one part of the former to two of the latter.

A saturated solution of silver nitrate.

A solution of nitrate of mercury made by acting on 71·5 grammes of mercury with five times its weight of pure nitric acid (specific gravity 1420), heating until no nitrous fumes are given off, and, after evaporating to a syrupy consistence, adding water to 1 litre. This will precipitate 10 grammes of urea, and it is therefore of such a strength that 1 cubic centimetre is equal to 0·01 gramme urea. Before employing it, it should be tested, by the process about to be described, with a solution of urea of known strength.

A solution of carbonate of sodium of the strength of 1 to 20.

The method of procedure is as follows:—To 20 cubic centimetres of urine add 20 cubic centimetres of the solution of barium salts. Mix them thoroughly and filter. This removes all the phosphates and sulphates. To the filtrate add 20 cubic centimetres of the solution of silver nitrate, and after mixing the fluids again filter. By this means the chlorides are separated and removed, and the filtrate is ready for estimation. Of this, put 30 cubic centimetres, equal to 10 cubic centimetres of the original urine, in a glass vessel, and place some of the solution of sodium carbonate upon a porcelain plate. The solution of nitrate of mercury is now to be added slowly from a graduated burette and the urine stirred carefully. After the first 5 or 6 cubic centimetres of the test solution have been added, place a drop of the urine in the solution of sodium carbonate on the porcelain plate, and, if there is no change of colour, add 1 cubic centimetre of the test solution, and again place a drop of urine on the sodium carbonate solution. This is to be continued, testing after the addition of each cubic centimetre of the mercury solution, until a yellow colour is observed, showing that all the urea has been precipitated, and the number of cubic centimetres employed will give the number of centigrammes of urea in 10 cubic centimetres urine.

If, for example, 15 cubic centimetres are required, $15 \times 0{\cdot}01 = {\cdot}15$ gramme of urea in 10 cubic centimetres of urine, and if 1600 cubic centimetres form the daily quantity of urine, then—

$$10 : 1600 :: {\cdot}15 : 24$$

or the quantity of urea is 24 grammes in that period.

The acidity of the mercury solution has considerable influence on the reaction, and the mercuric nitrate must be prepared with as small an excess of acid as possible. It has been shown by Pflüger that the alkalinity of the mixture also interferes with the process. If the urine is alkaline much more of the mercury solution is necessary to bring about the development of the end-reaction. It has further been pointed out by the same author that if the solution of mercuric nitrate is rapidly added to the urine, a much larger quantity is required than if it be added drop by drop.

As most of the other nitrogenous substances found in urine enter into combination with the mercury solution, and as many drugs, such as iodides, salicylates, and benzoates, which are excreted by the kidneys, also combine with it, the method of Liebig cannot be regarded as accurate; in fact, as Salkowski points out, it is only available for normal urine. It is at the same time a much more troublesome process than that which has now to be described.

Volumetric analysis by decomposition may be performed by means of a freshly prepared solution of hypobromite of sodium or chlorinated soda. The former reagent is to be preferred. It must not be forgotten that other nitrogenous bodies besides urea are broken up by means of this process, but it is also to be remembered that by its means there is a deficiency in the volume of nitrogen evolved as tested with a pure solution of urea.

For this process a special apparatus is necessary. It consists of—

A flask furnished with an indiarubber stopper, through which a glass tube is inserted.

A basin of water into which the flask may be put.

A small test-tube graduated to 5 cubic centimetres.

A tube about 40 centimetres in length, with a calibre of about 1·75 centimetres.

This tube is drawn out at one end to form a smaller tube connected with the flask by a piece of indiarubber tubing. The tube, placed with the small end uppermost has a zero mark upon it about half an inch below the commencement of that part with the smaller calibre. From this zero mark downwards a space equal to 55 cubic centimetres is divided into 30 parts of equal capacity. A volume of nitrogen filling one of these divisions and evolved from 5 cubic centimetres urine is exactly equivalent to 1 per cent. of urea, or 1 gramme of urea in 100 of cubic centimetres of urine.

A glass cylinder, to be filled with water, in which the graduated tube is held by means of a clamp.

A graduated measure with a mark for the quantity of bromine, which is 1·3 cubic centimetres, and another for the solution of caustic soda, which is 13·7 cubic centimetres, the total bulk of the solution of hypobromite of sodium employed being 15 cubic centimetres.

One of the usual forms of this apparatus—that of Russell and West—is figured below.

The solutions required are—

Bromine.

A solution of caustic soda containing 100 grammes of caustic soda in 250 cubic centimetres of water.

The process is to be carried out in the following manner:—

Measure out the bromine, add the caustic soda solution, and after mixing them well pour the fluid into the flask.

Measure out 5 cubic centimetres urine in the small test-tube, and by means of a forceps lower it into the flask, taking care that none of it spills into the hypobromite solution. Fix the stopper firmly in the flask, which will then be in communication with the graduated tube.

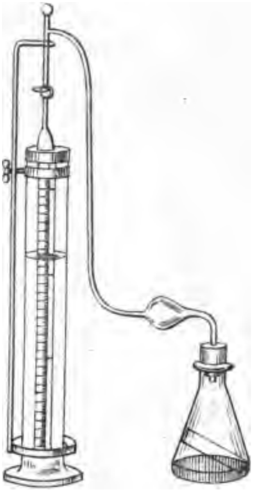

FIG. 71.—Ureometer of Russell and West.

See that the water in the glass cylinder and in the graduated tube stands at the zero mark on the latter.

Leave the apparatus for ten minutes until the temperature becomes uniform throughout the system, and then incline the flask so that the urine flows from the small test-tube into the flask, gently shaking so as thoroughly to mix the two fluids. A considerable amount of effervescence ensues with the development of heat, and the water sinks in the graduated tube from the development of nitrogen. The completion of the reaction will be known by the cessation of effervescence.

During the process the flask must be kept cool with the cold water in the basin. When the reaction is at an end, the water is to be brought to the same level in the tube and outside of it. The index is then to be read, and the number of spaces occupied by nitrogen gives the per-centage of urea expressed for each space as 0·1 gramme urea in 100 cubic centimetres urine.

If, for instance, 15 spaces are filled by the gas, the urine contains 1·5 per cent. of urea, and if there be 1600 cubic centimetres passed in twenty-four hours, then—

$$100 : 1600 :: 1{\cdot}5 : 24$$

or the quantity of urea is 24 grammes in that period.

This process, as was referred to above, falls short of the absolute amount of urea by 8 per cent.; that is, at 0° C. and 760 millimetres pressure, 1 gramme of urea evolves only 342·9 cubic centimetres of nitrogen, instead of 372·7 cubic centimetres, which is the theoretical amount. Knowing this fact, it is perfectly easy to calculate the exact quantity of urea contained in any given urine.

The results obtained by the hypobromite process require to be corrected for pressure and temperature in order to be quite accurate. If V be the volume desired at 0° C., and 760 millimetres pressure; v the volume read off; P a pressure of 760 millimetres of mercury; p the atmospheric pressure of the room; T the absolute temperature (zero centigrade $= -273°$); t the temperature of the room (expressed in degrees centigrade $+ 273$); then—

$$VPt = vpT;\ v = \frac{VPt}{pT};\ \text{and}\ V = \frac{vpT}{Pt}$$

If the volume of nitrogen evolved under a barometric pressure of 755 millimetres of mercury, and at a temperature of 10° C., fills twenty spaces of the ureometer, then—

$$V = \frac{20 \times 755 \times 273}{760 \times 283} = 19{\cdot}11$$

that is, at 0° C., and under 760 millimetres of mercury, the number of spaces filled would be 19·11, from which the amount of urea may be calculated.

As already mentioned, 343 cubic centimetres of nitrogen at 0°C. and 760 millimetres of mercury are equal to 1 gramme of urea.

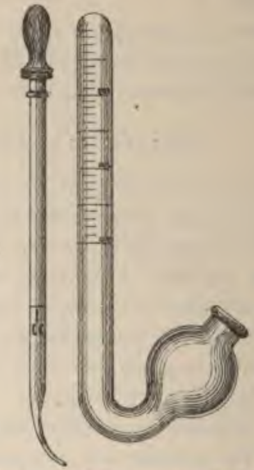

FIG. 72.—Ureometer of Doremus and Thursfield.

A very simple and exceedingly cheap modification of this process has lately been introduced by Doremus and Thursfield. The apparatus consists of a graduated tube and a pipette, which are figured above. The tube is closed at the upper end, curved at the lower end, and terminates in a bulb. The upper part is graduated, each division indicating 0·001 gramme of urea in 1 cubic centimetre of urine, and a mark at the bend shows how much of the reagent is required. The pipette has a mark indicating 1 cubic centimetre, and an indiarubber bulb for suction. In order to use the apparatus, pour the solution of sodium hypobromite up to the mark, filling the graduated arm, and add sufficient water to fill the bend and lower part of the bulb. Draw the urine into the pipette as far as the mark on it. Pass the pipette into the tube as far as the bend, and squeeze the indiarubber bulb so as to eject the urine, which rises through the hypobromite of sodium solution, evolving the gas which rises to the upper part of the tube. Each division indicates 0·001 gramme of urea from the 1 cubic centimetre of urine, and the per-centage is obtained by multiplying the result by 100.

Certain other nitrogenous substances found in urine are decomposed by the hypobromite of sodium, and this chemical reaction is apt to vitiate the result.

Uric acid is the most important disturbing element, and yields 47·7 per cent. of its nitrogen. In order to obviate this source of error the uric acid may be precipitated by basic acetate of lead, or it may be estimated by such a rapid process as that of Haycraft, described below, and the error caused by its presence corrected as follows:—

In each gramme of uric acid there is 0·333 of a gramme of nitrogen; but only 47·7 per cent. of this can be collected, so that from each gramme of uric acid only 0·1588 gramme of nitrogen can be obtained. But 0·1588 of a gramme of nitrogen at 0° C. and 760 millimetres in pressure is equal to 127 cubic centimetres, or is equivalent to 0·34 gramme of urea. From this it is easy, knowing the quantity of uric acid, to correct our urea estimation as far as this particular nitrogenous matter is concerned.

Hippuric acid, from the recent researches of Noël Paton,[1] gives off absolutely no nitrogen under the action of sodium hypobromite.

Kreatinin according to Schleich and Falck yields 37·5 per cent. of its nitrogen when acted on by the hypobromite solution, but as the quantity of kreatinin is small this source of fallacy is not great.

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[1] *The Practitioner*, vol. xxxviii., p. 168, 1887.

Tyrosin, as Noël Paton has proved, when in a fresh solution, evolves 8·5 per cent. of its nitrogen. This is not to be regarded, however, as an important possibility of error, for the amount of nitrogen in tyrosin is small, and the substance itself is only present in certain cases and in small proportions.

Leucin has been shown by Noël Paton to give off absolutely no gas when acted on by sodium hypobromite.

If glucose is present in the urine it increases the amount of nitrogen by 6 or 7 per cent.,—that is, it causes almost the whole of the nitrogen of the urea to be evolved. It appears to do so by the great heat which is produced by the mixture of glucose and the hypobromite solution, and hence the precautions in regard to the cooling of the apparatus during the process.

In carrying out the volumetric method with sodium hypobromite, Noël Paton urges attention to the following precautions :—

(1.) That the same apparatus and same strength of hypobromite solution be always used, and that they should be first tested upon a standard solution of urea.

(2.) That the hypobromite solution should be freshly prepared each day.

(3.) That the urine, when concentrated, should be diluted to a specific gravity of between 1010 and 1020.

(4.) That full time be allowed for the temperature throughout the apparatus to become uniform before and after the decomposition.

(5.) That the volume of gas be corrected for temperature and pressure.

(6.) That throughout the decomposition the temperature of the flask be kept low by submerging it in a large basin of water at the temperature of the room.

With these precautions he claims for the hypobromite method greater accuracy than Liebig's method can afford, while the ease with which it may be carried out renders it of very special value in clinical investigations.

The daily quantity of urea is chiefly dependent on the amount of nitrogen taken in the food. This is a fact which should never be forgotten by the physician. It is markedly increased in all conditions accompanied by, or dependent on, greater activity of the metabolic processes, such as pyrexia amongst acute diseases, and diabetes mellitus amongst chronic. It is, on the other hand, diminished in Bright's disease, in many disorders of the hepatic functions, and, speaking generally, in wasting diseases.

Uric acid ($C_5H_4N_4O_3$) is thrown out of its combinations with bases by the addition of strong hydrochloric acid, and is then, on account of its insolubility, precipitated; it may in this way be procured for the purpose of examination. *It can be recognised* chemically by means of the murexide test, which is also applicable to the salts which it forms with alkalies and alkaline earths. This test is performed with crystals of these substances, or with the residue obtained by evaporating urine. It consists in the addition of a drop of strong nitric acid and subsequent heating to dryness, which gives a reddish-brown deposit on the porcelain capsule employed, and this, when touched with a glass rod which has been dipped into a strong solution of ammonia, at once assumes a crimson tint. If caustic potash is used instead of ammonia, a violet colour is obtained.

When separated out by the addition of hydrochloric acid, uric acid is found to be very insoluble, its solubility in cold water being only 1 in 15,000. It is a dibasic acid, and forms both neutral and acid salts with the metals of the alkalies and alkaline earths, all of which are much more soluble than the acid. Uric acid has a reducing action on the cupric tests for glucose, as will be described under that head. It is also precipitated by strong acids, and is apt to be a source of error in the cold nitric acid test for albumin as will be seen in the sequel.

Uric acid, when examined microscopically, is found in rhombic crystals of many different forms, some of which are shown in Fig. 73 and Fig. 74. Lozenge and oval shaped plates and spikes or rods are the most common forms of crystal; these are frequently grouped together to form sheaf-like or fan-shaped bodies, along with stellate and rose-formed collections.

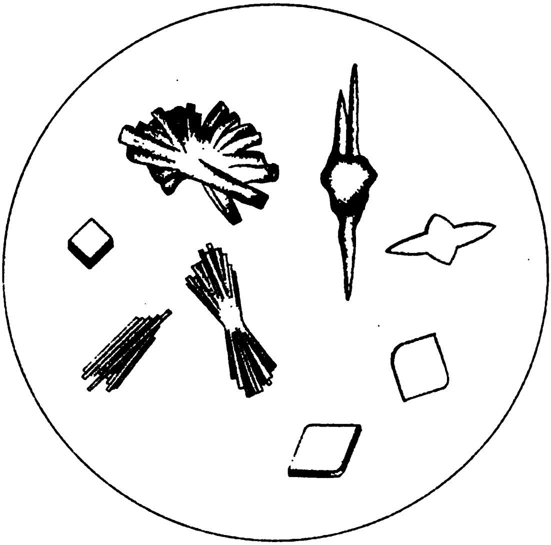

FIG. 73.—Uric acid.

Uric acid may be estimated volumetrically by precipitating it by the addition of strong acid and weighing the deposit.

This quantitative estimation may be performed by taking 200 cubic centimetres of urine, and if its specific gravity is below 1015, concentrating it by evaporation until this rises to 1020. To this urine add 20 cubic centimetres strong hydrochloric acid, and thoroughly mix, setting aside the mixture in a urine glass for twenty-four hours. The upper part of the fluid is to be decanted off, and the lower portion to be filtered through a piece of filtering paper that has been thoroughly dried and afterwards weighed. The filter with its deposit is to be washed with water acidulated with hydrochloric acid, and afterwards with alcohol; it is then to be dried in an air bath at a temperature of 100° C., and the difference in weight gives the quantity of uric acid in 200 cubic centimetres urine, from which the amount of the daily secretion may easily be determined. If, for example, the filter originally weighs 0·25 gramme, and along with the uric acid 0·37 gramme, the quantity of uric acid in 200 cubic centimetres urine is 0·12 gramme, and if the daily amount of urine is 1400 cubic centimetres, the weight of uric acid secreted in twenty-four hours will be obtained thus:—

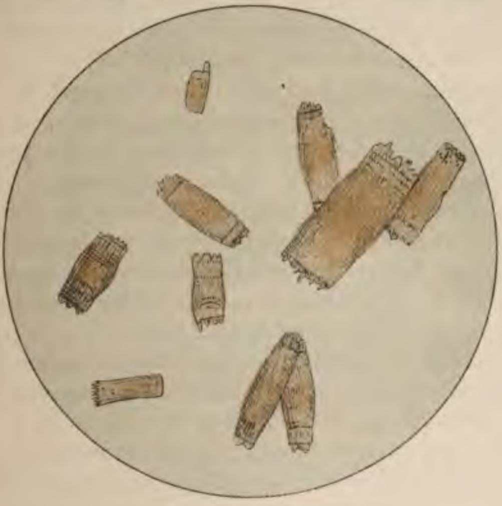

FIG. 74.—Uric acid.

200 : 1400 : : 0·12 : 0·84

and the quantity passed in that period is therefore 0·84 of a gramme.

Uric acid may be rapidly estimated by the method introduced by Haycraft.[1] This method is based upon the fact that urate of silver is insoluble in ammonia water. On adding a solution of silver nitrate to a solution of acid urate of sodium, instead of a precipitation of silver urate, there is an immediate reduction, the black precipitate not re-dissolving in ammonia water. If the solutions be previously rendered ammoniacal, a white gelatinous precipitate of the urate at once forms. The silver, however, becomes partially reduced before it is possible to collect and wash it, but the previous addition of bicarbonate of sodium prevents this reduction. The same obtains with the acid urate of sodium normally present in urine, the chlorides and phosphates remaining in solution, the urate of silver alone falling on the addition of the ammoniacal nitrate. In order to estimate the silver in this precipitate of the urate, the latter is collected in a filter, and washed with distilled water, and, taking advantage of its ready solubility in nitric acid, it is dissolved in that reagent.

This process is carried out in the following manner :—

Solutions required.—1. Centinormal ammonic sulphocyanate. Dissolve about 8 grammes of crystals in a litre of water, and adjust it to decinormal silver solution. Dilute with 9 volumes of water. One cubic centimetre is equivalent to 0·00168 of a gramme of uric acid.

2. A saturated solution of iron alum.

3. Pure nitric acid (20-30 per cent.). Dilute the commercial acid, boil and preserve from light in a blackened flask.

4. Strong ammonia.

5. Ammoniacal silver solution. Dissolve 5 grammes of nitrate of silver in 100 cubic centimetres water, and add ammonia, until the solution becomes clear.

Method.—Measure off 25 cubic centimetres of urine in a pipette, and place it in a small beaker, with about 1 gramme of bicarbonate of sodium. Add 2 or 3 cubic centimetres of ammonia, which will produce a precipitate of ammonia-magnesium phosphate. On adding 1 to 2 cubic centimetres of the ammoniacal silver solution, the uric acid falls as a white gelatinous precipitate of urate of silver.

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[1] *British Medical Journal*, 1885, vol. ii. p. 1100.

This is collected on the asbestos filter, and carefully washed until the washings give no trace of silver with a drop of weak hydrochloric acid. The urate is then washed through the filter by the aid of a few cubic centimetres of the nitric acid, and the silver in this solution estimated by Volhard's method.

Add a few drops of the saturated solution of iron alum, which is the indicator, and drop in the centinormal solution of ammonic sulphocyanate. A white precipitate will form, together with a transient reddish coloration, which latter becomes permanent when the process is at an end.

It is easy to calculate the uric acid, which will be the number of cubic centimetres of the sulphocyanate used multiplied by 0·00168.

If the urine contains albumin, this should previously be removed. If uric acid or urates are present in such quantity as to cause turbidity, the secretion should be warmed and diluted.

The quantity of uric acid secreted is usually found to vary in direct proportion to the increase or decrease of urea. It is increased in all febrile conditions. During an acute attack of gout it is almost invariably diminished in quantity, but there is a considerable increase above the normal after the paroxysm has passed off.

Crystalline uric acid is spontaneously deposited from fresh urine in certain highly acid conditions, but this occurrence has but little clinical importance, except from a therapeutic point of view.

Urates.—As above mentioned, uric acid forms salts with metals of the alkalies and alkaline earths. The only urates commonly met with, however, amongst the products of renal activity are salts of sodium and ammonium. Urates may be detected by means of the murexide test above described, and, as they are decomposed by hypobromite or hypochlorite of sodium, they give rise to part of the nitrogen evolved in the volumetric analysis of urea as conducted by the decomposition process.

Deposits of urates are found in two forms, amorphous and crystalline.

Amorphous urates form a finely granular deposit of variable colour—cream, fawn, orange, red, or pink—depending on the amount of the colouring matters of the urine. This deposit is usually thrown down at once as soon as the urine has cooled, and along with the sediment at the bottom of the glass there is a fine film upon the sides. The presence of this thin film is characteristic of urates, and serves to distinguish them from all other deposits.

The application of heat to urine containing urates causes them to dissolve, and as they form the only urinary deposit which disappears on heating, the use of the spirit lamp gives an easy means of recognising them.

Amorphous urates have not a fixed chemical composition; the uric acid is combined with several bases, sodium, ammonium, potassium, and calcium, in variable proportions, and deposits of this nature are to be regarded as consisting of mixed urates.

Microscopically amorphous urates are seen as a mass of granules, larger or smaller, according to circumstances.

Amorphous urates are precipitated when their proportion to the quantity of urine rises, and a deposit is always present if the amount of the urine is diminished from any cause. An increase of acidity also favours the formation of a deposit, and a low temperature of the atmosphere, by rapidly cooling the urine, tends likewise to the same result.

The presence of a deposit of amorphous urates has no great clinical significance. Such a deposit is formed very frequently in cold weather, and after profuse perspiration or violent exercise. It is almost invariably present in any febrile condition, and it is a common occurrence in digestive disorders. When a deposit of this nature is frequently or constantly to be found in any urine, it should lead to a thorough examination of every system, in order to determine the cause of the altered metabolism giving rise to the condition.

Crystalline urates.—Deposits of urates in crystals are much less common than the amorphous form just described. Such deposits are composed of urate of sodium and urate of ammonium.

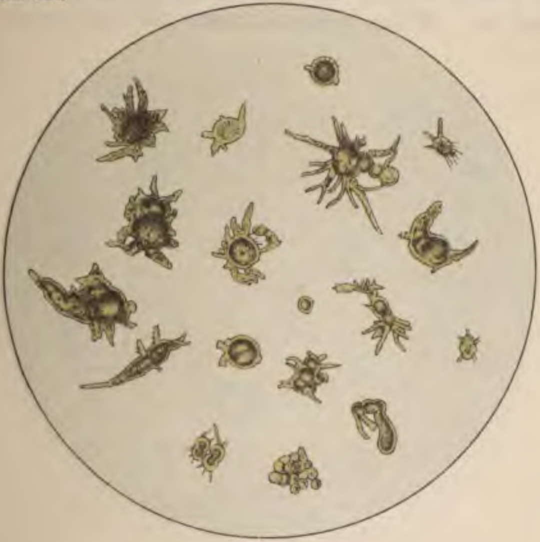

FIG. 75.—Urate of sodium.

Urate of sodium forms a greyish or yellowish deposit, and when examined microscopically is found, as in Fig. 75, to assume various irregular outlines, with spines projecting from them. As a urinary deposit, this salt is never found in the beautiful acicular crystals which form gouty concretions.

When found in the urine, crystalline urate of sodium usually points to a highly acid state of the secretion. It is most commonly found in gouty conditions and in the pyrexia of childhood. The main significance to be attached to this deposit is the circumstance that it is precipitated within the urinary passages, and is apt to cause various disorders in consequence of its presence.

Urate of ammonium, as has already been seen, is thrown down as a deposit during the alkaline fermentation of the urine. The precipitate is almost invariably white in colour. It is always accompanied by the phosphates previously referred to. When examined under the microscope it is found in opaque spherical masses, or in elongated dumb-bells. These latter frequently become united, and form crosses or stars.

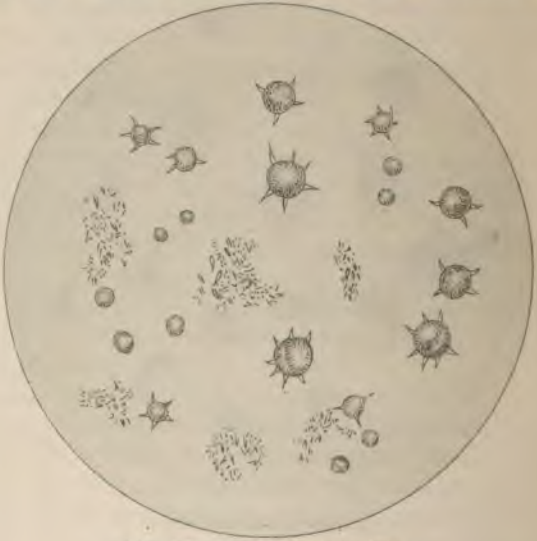

FIG. 76.—Urate of ammonium.

Urate of ammonium has in itself no clinical importance.

It is merely one of the phenomena caused by the alkaline fermentation.

Hippuric acid ($C_9H_9NO_3$) occurs in small quantities in healthy human urine, chiefly as a sodium salt, and is present in larger quantities in the urine of herbivorous animals. It is almost insoluble in cold water, but is much more soluble in hot water, and very easily dissolved by a solution of sodium phosphate. It is a monobasic acid, and forms precipitates of a buff colour with ferric salts. It is a crystalline substance, and when examined under the microscope is seen, as in Fig. 77, to consist of rhombic prisms.

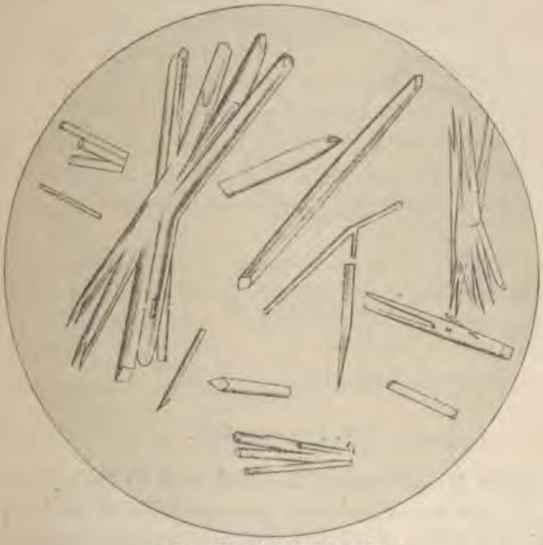

FIG. 77.—Hippuric acid.

The amount of hippuric acid in the urine of twenty-four hours is from ·88 to 1 gramme, or from 12 to 15 grains. In health it is greatly increased by the administration of benzoic acid, and by a vegetable diet, especially by the use of certain fruits.

In febrile conditions and in diabetes mellitus it is also increased.

Kreatinin ($C_4H_7N_3O$) is present in considerable relative proportions. It is a powerful base, able to drive ammonium out of its combinations, and may be recognised by its crystalline form, which belongs to the oblique rhombic system as shown in Fig. 78.

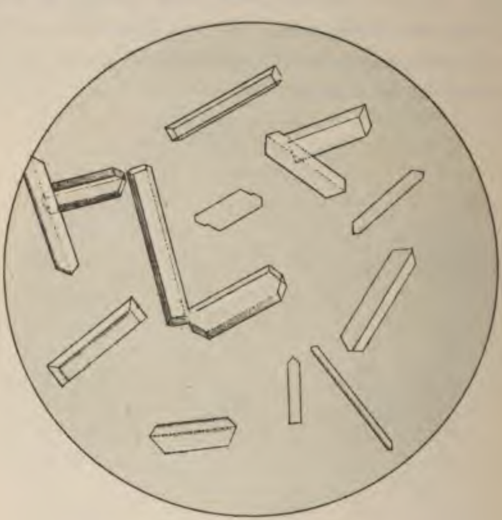

FIG. 78.—Kreatinin.

To detect the presence of kreatinin add to the urine a few drops of pale red solution of nitro-prusside of sodium, and then, drop by drop, a dilute solution of caustic potash, when a ruby-red colour is struck, varying in intensity according to the amount of kreatinin present. The colour soon passes into a straw-yellow tint.

The quantity of kreatinin in the urine of twenty-four hours varies in health from ·5 gramme to 1 gramme, or from 7 to 15 grains. It is derived from the kreatin of the muscles. The clinical significance of variations in the daily amount is at present uncertain; it is, however, increased in health by a meat diet and lessened by vegetable food, while it is increased by febrile conditions and diminished by renal disorders and all wasting diseases.

Xanthin ($C_5H_4N_4O_2$) occurs in extremely small quantities as a constituent of healthy urine, and may be recognised both by chemical reactions and microscopic appearances.

Oxalic acid ($C_2H_2O_4$) is normally present in the urine in minute quantities, combined for the most part with alkalies to form soluble salts. As the result of fermentation, and in certain disordered conditions, it makes its appearance as the insoluble oxalate of calcium, to be described under the heading of abnormal urinary constituents.

The quantity of oxalic acid is considerably increased by the use of sugar, starch, and many fruits. Disorders of digestion and derangements of the processes of tissue-change cause the appearance of a larger quantity, but the conditions in which an increased amount may be expected are as yet unknown.

Lactic acid ($C_3H_6O_3$) is not a normal constituent of fresh urine, but is formed during the process of acid fermentation. It may be detected after careful manipulation as a calcium or zinc salt.

The urine contains lactic acid in rickets, osteomalacia, and certain digestive disorders.

Pigments.—Of the normal organic constituents of the urine, the colouring matters, urobilin and indican, alone remain to be described.

Urobilin ($C_{32}H_{44}N_4O_7$) is constantly present in healthy urines, and MacMunn states that he has never failed to recognise it in normal conditions, although it may be absent in disease.

When separated from urine, urobilin is a dark reddish-brown powder, freely soluble in alcohol, ether, and chloroform, but less easily dissolved in water. Strong solutions have a reddish-brown colour, while weaker solutions assume a rosy hue. A solution in chloroform has an orange tint, and an alkaline solution is yellow, but turns red on the addition of acids. If it is dissolved in presence of ammonia, and a solution of zinc chloride is added, the solution has a rose colour and a beautiful green fluoresence is obtained.

To show the presence of urobilin in urine all bile pigment must be removed if any is present, the urine is to be rendered alkaline with ammonia, and filtered, and on the addition of a few drops of a solution of zinc chloride the greenish fluoresence above referred to makes its appearance.

By means of the spectroscope urobilin can be recognised without difficulty either in urine or in an acid solution. The spectrum shows a dark absorption band extending from a little to the right of *b* to a little beyond *F*. This band disappears on the addition of ammonia, but returns on the addition of a solution of chloride of zinc, although somewhat more to the left than the original position.

The quantity of urobilin is increased in febrile conditions, and certain affections of the digestive system, while it is diminished in renal diseases. Changes in the relative quantity have been referred to in the section on the colour of the urine.

Amongst organic sulphur-containing bodies, *Indican* ($C_8H_6NKSO_4$) or indoxyl-sulphate of potassium, containing sulphuric acid in organic combination, is almost always, if not quite constantly, present in healthy urine.

When separated from urine, it occurs in the form of bright tables and plates which are easily dissolved in water, but less soluble in alcohol. An acid solution turns blue on the addition of a few drops of a solution of calcium chloride.

To demonstrate its presence in urine, add to a small quantity of urine some hydrochloric acid and two or three drops of nitric acid. The mixture on heating assumes a reddish violet colour, and crystals of indigo blue and indigo red are formed.

Another method is as follows:—

Take five cubic centimetres of strong hydrochloric acid and heat it to about 70° C. Float on this an equal volume of urine, free from albuminous or biliary substances, and a ring will be formed which will be blue or violet, according as more or less indican is present. Mix the acid and urine thoroughly and add solution of calcium chloride until a greenish tint is produced. Shake the solution with 10 cubic centimetres of chloroform, which will separate out with a blue or violet colour.

Indican gives no spectroscopic phenomena when in solution in the urine, but the chloroform solution just mentioned, when examined by the spectroscope, gives, as MacMunn shows, two dark absorption bands in the spectrum, one a little before *C*, and the other beyond it.

The amount of indican excreted daily is believed to vary from 0·004 to 0·02 grammes. A flesh diet increases the amount, and the quantity is also greater in many digestive disorders, especially in obstruction of the bowels, and in cases where there is undue delay in the passage of the food through the intestines.

The *inorganic constituents* to which it is necessary to call attention are chlorides, sulphates, and phosphates.

Hydrochloric acid (HCl) is passed out in the urine chiefly in combination with sodium, but also to a smaller extent combined with potassium and ammonium. As the chlorides are soluble they never form urinary deposits, but if a few drops of urine are evaporated in a watch-glass octahedral crystals and rhombic plates of chloride of urea and sodium are formed.

Chlorides may be detected by the addition of a solution of nitrate of silver (1 to 8) which throws down a white precipitate of silver chloride, but, as phosphates also form new compounds with solutions of nitrate of silver, a few drops of nitric acid should be added, which prevents the precipitation of phosphate of silver.

Quantitative estimation of chlorides can be carried out by means of Mohr's nitrate of silver method.

The solutions required are :—

A solution of nitrate of silver prepared by dissolving 29·075 grammes of fused silver nitrate in a litre of water. This solution is of such a strength that 1 cubic centimetre is equivalent to 0·01 gramme of sodium chloride, or 0·006 gramme of chlorine.

A saturated solution of neutral potassium chromate to show the end-reaction.

To conduct the process take 10 cubic centimetres of the urine after it has been filtered, place it in a platinum crucible, add a gramme of pure nitrate of potassium, and evaporate to dryness. Expose the residue to a strong heat to consume the organic substances, and dissolve the white saline residue in enough of distilled water to make 100 cubic centimetres. Add a few drops of the solution of potassium chromate, stir the mixture well, and gradually add from a burette the silver nitrate solution, until a reddish tinge shows itself around the white precipitate formed, when each drop of the silver solution falls into the urine. The red tinge is caused by the formation of silver chromate, and its appearance shows that all the chlorine has been precipitated.

The number of cubic centimetres of the silver nitrate solution employed gives the amount of sodium chloride (and hence of chlorine) in 10 cubic centimetres of urine; from which the calculation of the daily excretion is easy. If, for instance, 5 cubic centimetres of the solution are used, then $5 \times 0{\cdot}01 = 0{\cdot}05$ gramme of sodium chloride, or $5 \times 0{\cdot}006 = 0{\cdot}03$ gramme of chlorine in 10 cubic centimetres. And if the daily amount of urine is 1600 cubic centimetres, then—

$$10 : 1600 :: 0{\cdot}05 : 8$$

the amount of sodium chloride is 8 grammes, or—

$$10 : 1600 :: 0{\cdot}03 : 4{\cdot}8$$

the quantity of chlorine is 4·8 grammes in twenty-four hours.

The quantity of chlorides excreted in health varies almost exactly in direct proportion to the quantity taken with the food, and may be said to lie within the limits of from 5 to 8 grammes daily.

In disease the quantity may depart greatly from the normal. Ague, during the cold and hot stages, causes a considerable increase, but this is the only febrile affection in which there is any augmentation. In every other disease attended by pyrexia the amount is diminished, especially in acute pneumonia, which may show a total disappearance of chlorides, as well as in diseases accompanied by exudations, such as pleurisy with effusion. This is probably due to the fact that the fluid poured out into the serous cavity is very rich in chlorides.

Sulphuric acid (H_2SO_4) appears in the urine as preformed sulphuric acid in organic combination (indican), or as inorganic compounds with potassium and sodium, and as its compounds with these metals are very soluble, no urinary deposits of this nature are to be met with.

Sulphates may be detected by means of a solution of barium chloride or nitrate, which throws down a white precipitate of barium sulphate insoluble in acids.

Quantitative estimation of sulphuric acid is carried out by means of a solution of barium chloride, and the end-reaction is determined by testing the mixed solutions with a solution of potassium sulphate.

The following are the solutions required :—

A solution of barium chloride prepared by dissolving 30·5 grammes of the dry salt in a litre of water ; of this solution 1 cubic centimetre is equivalent to 0·01 of a gramme of anhydrous sulphuric acid.

A solution of potassium sulphate prepared by dissolving 21·775 grammes of the salt, dried at 100° C., in a litre of water : 1 cubic centimetre of this solution contains 0·01 of a gramme of the anhydrous sulphuric acid.

The process is carried out as follows :—

Take 50 cubic centimetres of urine, add ten drops of hydrochloric acid, heat, and when boiling add by means of a burette 3 or 4 cubic centimetres of the barium solution. This is done to decompose the combined sulphuric acid substances. On removing the heat and allowing the precipitate to fall, if the fluid rapidly clears, add 1 or 2 cubic centimetres more of the barium solution, again heat, filter a few drops of the mixture into a test-tube and add a small quantity of the barium solution to it. If a precipitate is formed return the contents of the tube to the vessel, and gradually add more of the barium solution, from time to time testing a few drops in a tube as just described. When the barium solution ceases to form a precipitate, filter a small quantity of the urine into a test-tube, and add a few drops of the potassium sulphate solution. If a faint precipitate appears, then only a slight excess of the barium solution has been employed; if a dense precipitate is formed, a considerable excess has been added. From the results of the reaction it is easy to go over the steps of the process a second time, and to obtain with accuracy the end-reaction, *i.e.*, till no precipitation occurs on the addition of the barium solution, and only a faint turbidity with the potassium sulphate.

This process gives the amount of anhydrous sulphuric acid in 50 cubic centimetres of urine, and it is easy to arrive at the daily excretion by calculation from this. If, for example, 8 cubic centimetres of the barium solution are required, then $8 \times 0{\cdot}01 = 0{\cdot}08$ gramme in 50 cubic centimetres of urine, and if the daily quantity of urine is 1600 cubic centimetres, then—

$$50 : 1600 :: 0{\cdot}08 : 2{\cdot}56$$

the quantity of anhydrous sulphuric acid passed in twenty-four hours is 2·56 grammes.

The daily average of sulphuric acid passed in the urine is from 2 to 4 grammes. The quantity of sulphates is increased by a meat diet, and by the use of certain vegetables which contain sulphur. With the exception of plants containing sulphur, a vegetable diet lessens the quantity of sulphates excreted. The sulphates are increased by anything that hastens the rapidity of tissue change.

In all feverish conditions the amount is increased, especially in acute rheumatism and in acute affections of the brain and meninges.

Phosphoric acid (H_3PO_4) is the only acid of phosphorus which occurs in the urine, and this tribasic acid unites in various proportions with metals of the alkalies to form *soluble*, and with metals of the alkaline earths to form for the most part *insoluble* phosphates. One compound with an alkaline earth metal is soluble,—the primary magnesium phosphate, in which only one atom of hydrogen is replaced by the metal.

Alkaline or *soluble phosphates* are constantly found in the urine, in the form of acid salts of sodium and potassium (H_2NaPO_4 and H_2KPO_4). They give the urine its normal acidity, and are derived from the neutral phosphates of the blood. Ralfe has given an extremely ingenious explanation of the probable mode of their origin from the blood.

Earthy or *insoluble phosphates* only appear in neutral or alkaline urine, and are composed of several different substances.

Calcium phosphate (Ca_3, $2\ PO_4$), in which the three atoms of hydrogen in phosphoric acid are replaced by calcium, is found in alkaline urine, in the form of amorphous granules, or less commonly of acicular crystals, sometimes aggregated together as stellar masses. The amorphous deposit might possibly be mistaken for a sediment composed of urates, and perhaps the crystals might be taken for uric acid on careless inspection. If it is borne in mind, however, that the urine from which the deposits are obtained is alkaline, this fact will point to the strong probability that the sediment is phosphatic, to which the addition of a small quantity of a dilute acid will dissolve the phosphates, whereas it would, on the other hand, have no effect on the urates or uric acid.

Another much less common phosphate of calcium is sometimes met with, in which only two atoms of hydrogen are replaced by the metal. It appears in the form of wedge-shaped crystals, sometimes united as sheaves or rosettes.

Magnesium phosphate (Mg_3, 2 PO_4), a salt in which all the hydrogen of the phosphoric acid is replaced by magnesium, is usually found in association with calcium phosphate, and is deposited in the form of tabular crystals.

These earthy phosphates may be thrown down as a sediment in alkaline urine without any absolute increase in the quantity of phosphates eliminated, and the alkalinity of the urine in such cases usually depends on an excess of alkaline bicarbonates. On the addition of an acid such urines give a brisk effervescence.

There may, on the other hand, be a considerable increase in the excretion of phosphates without any deposit, on account of the acidity of the urine, but on boiling or the addition of alkalies precipitation at once takes place.

Ammonio-magnesium phosphate ($NH_4Mg\ PO_4$, 6 H_2O), often called triple phosphate, is a salt in which all the hydrogen of the acid is replaced by ammonium and magnesium. It is only formed when the urine assumes the volatile alkalinity previously described. It occurs in two forms—more commonly as triangular prisms or knife-rest crystals, and more rarely as feathery crystals. Like the calcium and magnesium phosphates, the triple phosphate is soluble in dilute acids. It is nevertheless sometimes found as a deposit in urine which is slightly acid, for which circumstance no satisfactory explanation has yet been given.

The presence of phosphoric acid may be demonstrated by the addition of a solution of silver nitrate, which throws down a yellow precipitate, soluble in ammonia and acids, or by the addition of a solution of ammonium molybdate in nitric acid and water, which also gives a yellow precipitate. On boiling urine which contains a considerable quantity of phosphates there may be, even when it is acid, a distinct cloud or turbidity, which disappears on cooling and on the addition of a dilute acid. The usual explanation of this fact is that the heat dissipates the carbonic acid of the urine which is believed to keep the calcium phosphate in solution. This does not explain the disappearance of the cloud on cooling, and it seems more probable that the cause of these appearances is, as Smith has suggested, an alteration of the proportions in which the alkaline earth bases unite with the phosphoric acid. On the addition of an alkali to the urine the earthy phosphates are precipitated, calcium phosphate as a rule in an amorphous form, and magnesium phosphate as a fine crystalline deposit. It must be borne in mind that when the urine is alkaline, phosphates are apt to be precipitated in the bladder, and in this case the first part voided is clear, while that which is passed towards the end of micturition is thick and muddy.

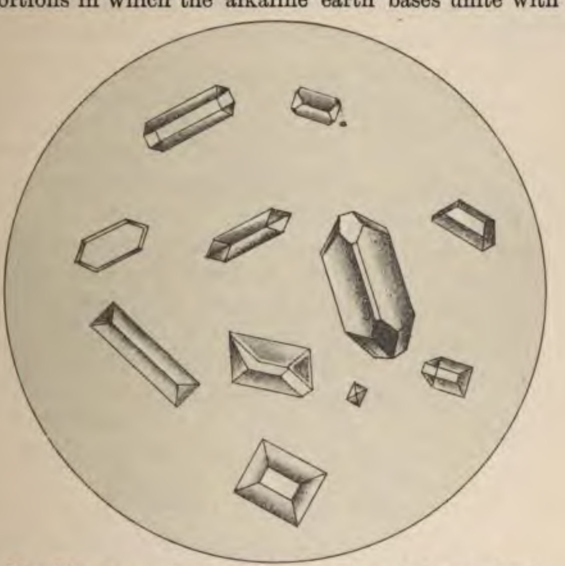

FIG. 79.—Ammonio-magnesium phosphate (prismatic form).

Quantitative estimation of phosphates is effected by means of an acid solution of uranium nitrate, which precipitates an insoluble phosphate of uranium; and the completion of the process is known by the fact that the nitrate gives a reddish-brown colour with ferrocyanide of potassium, while the phosphate causes no change in colour. An acid solution of acetate of sodium is employed in the process in order to make sure of the entire precipitation of the uranium phosphate.

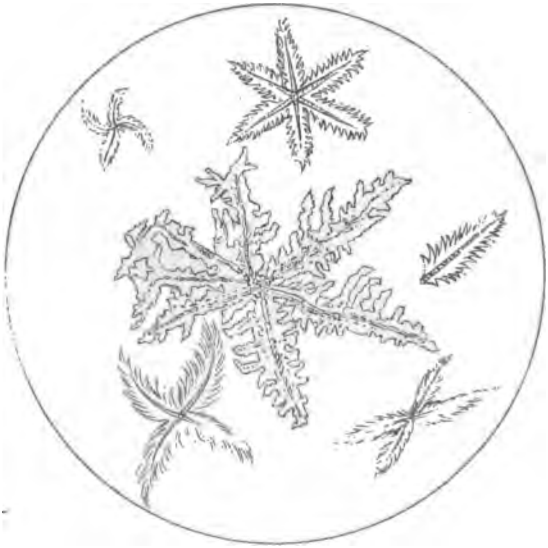

FIG. 80.—Ammonio-magnesium phosphate (feathery crystals).

The following are the solutions required:—

Solution of uranium nitrate prepared by dissolving 33 grammes of yellow oxide of uranium in nitric acid of 1200 specific gravity, adding water to 1100 cubic centimetres, and adjusting, by operating on a solution of sodium phosphate of known strength, the solution to such a strength that 1 cubic centimetre is equivalent to 0·005 gramme of anhydrous phosphoric acid.

Solution of acetate of sodium prepared by dissolving 100 grammes of the salt in 100 cubic centimetres of dilute acetic acid, and adding water to 1 litre.

A saturated solution of ferrocyanide of potassium.

To conduct the process take 50 cubic centimetres of urine, add 5 cubic centimetres of the sodium acetate solution, and heat. Add by means of a burette the uranium nitrate solution, stirring meanwhile until no precipitate is formed, and when this point is reached place a drop of the mixture on a porcelain plate along with a drop of the ferrocyanide solution. If the characteristic reddish-brown colour appears, add another cubic centimetre of the uranium nitrate solution, stir, and again test with the ferrocyanide of potassium solution. Continue this until no colour is given, which marks the end-reaction. The number of cubic centimetres of the uranium nitrate solution required represents the quantity of phosphoric acid in 50 cubic centimetres of urine, from which the daily amount may be calculated. For instance, if 14 cubic centimetres are used, $14 \times 0{\cdot}005 = 0{\cdot}07$, and if the daily amount of urine is 1600 cubic centimetres, then

$$50 : 1600 :: 0{\cdot}07 : 2{\cdot}24$$

the daily excretion of phosphoric acid is 2·24 grammes.

This process gives the total amount of phosphoric acid, whether combined with alkalies or alkaline earths.

The usual quantity of phosphoric acid which is excreted daily is from 2 to 4 grammes. It varies considerably with the nature and quantity of the food and the state of the nutritive processes. In disease it is increased in certain nervous affections, in the early stages of phthisis, in diabetes mellitus, and in leukæmia, while it is diminished in most acute diseases, in rickets, in gout, in rheumatism, and in renal diseases. While the total amount of phosphates is not greatly altered or slightly lessened in osteomalacia, the quantity of earthy phosphates is notably increased.

The *abnormal substances* which may be present in the urine are numerous, but it is only necessary to devote attention to albumin, blood, bile, sugar, and acetone, with certain crystalline matters, such as leucin, tyrosin, and cystin, and some organised deposits, such as epithelial, mucus, and pus cells, and casts of the renal tubules.

Albumin.—The proteids most commonly found in urine are serum albumin, serum globulin, peptones and albumoses, the last two being intermediate products formed during the process of digestion. Serum albumin may, under certain circumstances, become changed into acid albumin or alkali albumin. This may take place spontaneously if the urine is highly acid or highly alkaline.

In testing for proteids the urine should be filtered if turbid. If very highly acid it should be rendered less so by liquor potassæ. If alkaline it should be made slightly acid by means of acetic acid.

All these proteid bodies are precipitated by a number of reagents, and the presence of such substances may thus be determined. Most of the reagents in common use cause a precipitate with more than one form of proteid, and these tests are only useful in so far as they show that such a substance is present, without determining its nature more exactly.

Several of these reagents are employed as "contact-tests," that is to say, the urine to be examined is either underlaid or overlaid with the reagent, and a cloud at the line of junction of the two fluids shows that some proteid body is present.

Amongst such tests may be mentioned—

(1.) *The Nitric Acid Test* (*Heller's*).—Half a drachm of strong nitric acid is placed in a test-tube, and the suspected urine is slowly floated over this, or the urine may be placed in the tube and the acid gently poured down the side so as to lie below it. A cloud of coagulation immediately above the junction shows that some proteid is present. This test coagulates both serum albumin and serum globulin as well as the albumoses. It does not coagulate peptones. If a resinous body is present in the urine it causes a cloud at the line of junction, while uric acid and urates give rise to a zone of coagulation somewhat higher up in the tube. In concentrated urines this test may produce crystals of nitrate of urea, occurring in large scales above the contact line. Clouds formed by resins, uric acid, urates and nitrate of urea are dissolved by heat, which does not cause the disappearance of the cloud produced by the proteids.

(2.) *The Brine Test* (*Roberts'*).—The reagent is made by adding a drachm of dilute hydrochloric acid to a pint of water and saturating the solution with common salt, and it is employed in the same way as the nitric acid test, by underlaying the urine with it. The test coagulates the same proteids as are precipitated by the last test, as well as resinous bodies.

(3.) *The Mercuric Chloride Test* (*Tanret's*).—The solution employed in this test is made by mixing 1·35 grammes of perchloride of mercury, 3·32 grammes of iodide of potassium, 20 cubic centimetres of acetic acid, and 64 cubic centimetres of water. It is used in the same way as the last, by overlaying it with urine which must be strongly acid. It coagulates all the proteid bodies above mentioned.

(4.) *The Sodium Tungstate Test* (*Oliver's*).—This test is performed with a solution made by mixing equal volumes of a saturated solution of tungstate of sodium and of a saturated solution of citric acid, and adding to the mixture as much water as the united bulk of these solutions. The reagent is employed in a manner similar to that in which the former tests are used, and the results are the same as in the case of the mercuric chloride test.

(5.) *The Potassium Ferrocyanide Test* (*Pavy's*).—This test is conducted by acidulating the urine with acetic acid, and underlaying the mixture with a 1 to 12 solution of ferrocyanide of potassium. It precipitates all albuminous bodies present in urine, with the exception of peptones.

(6.) *The Picric Acid Test* (*Johnson's*).—A saturated solution of picric acid is floated upon the surface of the urine, and the tube is gently shaken in order to cause a slight mixture of the reagent with the urine. Cloudiness of the zone where the two fluids are mixed shows that one or other of the four proteids mentioned is present, unless the individual whose urine is under examination has been taking quinine or other alkaloid, which produces a substance coagulable by picric acid.

Other tests similar to these might be mentioned, but those which have just been described are the best of the kind in common use, and nothing need be said about the others.

The methods of distinguishing the proteids have been recently described in an admirable paper by Noël Paton,[1] whose procedure will be followed in the succeeding pages.

Serum albumin and serum globulin are coagulated by heat, and the application of heat is sufficient to determine that one of these substances is present, but is of no use in distinguishing one from the other.

The best method of employing the heat test is by slightly acidulating the urine with acetic acid and heating the upper part of the fluid in the test-tube. If serum albumin or serum globulin is present a cloud forms in the heated layer. Coagulation takes place just before the boiling-point is reached. It may present appearances varying from a slight haze to a milky cloud. If the cloudiness be faint, it may be most easily recognised if the tube is held up against a dark background.

Performed in this way there is no possible source of error. If the urine has not been acidulated a cloud may be formed by the deposition of phosphates, but this is at once dissolved on the addition of an acid. If the urine is very alkaline the serum albumin and serum globulin may have undergone the change into alkali albumin previously referred to, and this alkali albumin is not coagulable by heat. If the urine is highly acid, the serum albumin may be converted into acid albumin which does not coagulate on heating. A drop or two of liquor potassæ will change the acid albumin into serum albumin, and coagulation will occur with heat.

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[1] *The Edinburgh Medical Journal*, vol. xxxiv., p. 522, 1888.

If a precipitate occurs with the heat test it may be removed by filtration, and the filtrate can then be tested for peptones and albumoses. If no coagulation takes place, the original urine may be tested for these without filtration.

The filtrate or original urine is then to be tested for albumoses. This may be done by the cold nitric acid test, which gives a white cloud at the line of junction if albumose is present. A white precipitate on the addition of acetic acid and ferrocyanide of potassium, serum albumin and serum globulin having been excluded, is also significant of albumose.

If no albumoses can be detected in the urine it may be tested for peptones, but if albumoses are present they must be removed. This may be done by rendering the urine strongly acid with acetic acid, and adding powdered sulphate of ammonium until saturation occurs. This precipitates all proteids with the exception of peptone. They are to be removed by filtration, and the filtrate is then to be tested for peptone. This may be done by the picric acid test—either the saturated solution or Esbach's solution, to be mentioned presently, being available—which gives a cloud, disappearing with heat and returning again on cooling. Or this may be done by carefully neutralising the filtrate, after separating serum albumin and serum globulin, to bring down albumoses, and then testing with picric acid for peptones.

Serum albumin and serum globulin are usually associated together in any albuminous urine; they may be distinguished by the behaviour of the latter in neutral solution on the addition of magnesium sulphate. If the urine be rendered neutral or very faintly alkaline with liquor potassæ, and a saturated solution of magnesium sulphate be poured down the side of the test-tube, a white ring appears at the line of junction of the two fluids. By saturating the neutralised urine with sulphate of magnesium, all the serum globulin may be precipitated and after filtration and acidulation, a precipitate of serum albumin may be obtained on boiling when it is present.

Albuminometry, or the *quantitative estimation* of the important proteids, has within the last few years been much simplified, and the old gravimetric process, as well as the titration method by means of perchloride of mercury and iodide of potassium, has been entirely superseded for clinical purposes.

By means of *Esbach's process* the amount of proteids—which in most cases may be assumed to be composed of serum albumin and serum globulin—can be easily determined with considerable accuracy. The reagent employed is obtained by dissolving 10 grammes of picric acid and 20 grammes of citric acid in 800 or 900 cubic centimetres of hot water, and, after solution, adding enough water to make 1000 cubic centimetres or 1 litre when cold. It is employed in a special tube holding about 20 cubic centimetres, which is marked by a line and the letter "U" to indicate the quantity of urine to be used, and another line higher up with the letter "R," to show how much of the reagent is to be added to the urine. At the lower part of the tube are lines marked by the figures 1, 2, 3, 4, 5, 6, and 7, to indicate the result. The tube is provided with an indiarubber stopper.

The method of using the tube and reagent is as follows:—Fill the tube with the urine as far as the letter "U," and add the reagent until the fluid reaches the letter "R." Close the tube by means of the stopper, gently turn it upside down twice without shaking, and lay it aside for twenty-four hours, at the end of that period read off the height of the coagulation as shown by the figures at the lower end of the tube. The figure represents the number of grammes of proteids contained in a litre of urine.

In using this method it is of importance to attend to two points. The urine must, firstly, be quite acid. If it should happen to be alkaline or neutral, acetic acid must be added until it turns litmus paper to a bright red tint. The results, secondly, are more exact when the quantity of proteids is small, and in the case of highly albuminous urines it is well to dilute them so as to double or quadruple their bulk, taking care when recording the number of grammes in a litre to correct the result. If the amount is above ·6 per cent., or below ·05 per cent., the result is not accurate.

In order to determine the relative amount of serum albumin and serum globulin in any urine, all that it is necessary to do is to estimate by means of the Esbach process the total quantity of proteids (which as already mentioned may be considered to be solely composed of serum albumin and serum globulin) in the urine, and to estimate the quantity in it after complete precipitation of the serum globulin by shaking the neutralised urine with powdered magnesium sulphate. The result of this second estimation gives the amount of serum albumin, and the difference between it and the total quantity of proteids is the amount of serum globulin. Six days must elapse before the tube is read off, since the high specific gravity of the magnesium sulphate solution delays the precipitation of the albumin, and it may be necessary to shake the tube and determine the precipitation.

The *gravimetric method* of estimating serum albumin and serum globulin is performed by adding a few drops of acetic acid to a given quantity of urine, and boiling in a water bath for an hour. The urine is then filtered through a dried and weighed filter, which is afterwards carefully washed first with water, then with alcohol and ether, and lastly with alcohol, thoroughly dried in an air bath and weighed. The weight of the precipitate gives the amount of albumin and globulin in the quantity of urine.

The *clinical significance* of these proteids is still a matter of discussion. Serum albumin and serum globulin are almost invariably associated together, and it is almost beyond doubt that they transude by means of the glomeruli. It is almost equally certain that if the epithelium of the glomeruli is intact no transudation of proteids can occur, and the presence of serum albumin and serum globulin in conditions of apparent health—such as we find in what is called "functional albuminuria," "cyclical albuminuria," or "the albuminuria of adolescence"—may be taken as evidence that on account of some transitory disturbance of the kidney, or the blood-vessels, or the blood, there is a state of abnormal permeability of the glomerular epithelium. Albuminous substances are found in the urine as the result of febrile affections and toxic agents; venous stasis from heart and lung disease or pressure on the renal veins; temporary stoppage of a ureter; several chronic wasting diseases, such as anæmia, leukæmia, and tuberculosis; and certain diseases of the nervous centres, such as cerebral hæmorrhage. In these affections there may be no special lesions of the kidney. But the great cause of albuminuria is Bright's disease, and it is of the highest importance to be able to determine in any case by means of the whole of the symptoms whether there is evidence of renal disease or not.

Peptones have been found to be very common constituents of the urine in acute diseases, in the reabsorption of exudations, and when there is irritation of the urinary mucous membrane.

Albumoses used to be regarded as almost pathognomonic of osteomalacia, but recent researches would seem to indicate that they may be present in a number of different conditions.

Blood in the urine gives it a tint varying in degree with the quantity which is present. One part in two thousand gives a smoky tint, and one in five hundred produces a bright cherry colour. When blood is present the urine gives characteristic reactions with the tests for proteids, as serum albumin and serum globulin are always to be detected.

The colour of the urine is in most cases sufficient to determine the presence of blood, but it may be proved by spectroscopic, microscopic, and chemical examination.

Spectroscopic Examination.—If much blood is present, a specimen placed in a narrow test-tube and held in front of the slit of the chemical spectroscope may obscure the whole spectrum, but when diluted with a little water, some of the red and orange appears, and when still more diluted the green begins to show itself, while between the orange and green a dark space is seen. If the position of this absorption band is compared with Fraunhofer's lines it will be found to extend from beyond D towards the red side, to a point beyond E towards the violet side of the spectrum. On further dilution this broad band is found to be composed of two smaller bands; one near D being narrower but more strongly marked, the other near E being broader but less definite at its edges. By adding more water the bands can be narrowed, and that near E disappears, leaving the other to be faintly seen; and still further dilution finally removes it also. Reducing agents added to the urine cause the disappearance of these two absorption bands, which are replaced by one broader but less defined band midway between D and E. The addition of ammonium sulphide to urine containing blood will effect this change.

In what is known as hæmoglobinuria the spectrum of methæmoglobin is usually present.

Microscopic Examination.—Blood corpuscles retain their natural size and form for a considerable time in urine which is slightly acid, but at last become irregular in outline and cease to form rouleaux. When the urine is alkaline the form speedily alters, and the colouring matter makes its escape.

Chemical Examination.—The guaiac is the most satisfactory chemical test for blood in urine, but it must be remembered that it is not conclusive. The reagents required are a freshly prepared tincture of guaiac, which must be made with rectified spirit, the ammoniated tincture of the Pharmacopeia being useless for the purpose, and ozonic ether, that is, an etherial solution of hydrogen peroxide. The test is performed by placing a drachm of urine in a test-tube; adding a drop of the guaiac tincture, thoroughly mixing them, and gently shaking with as much ozonic ether as will equal the quantity of urine. When blood is present a bright blue colour is seen in the ozonic ether when it separates, or it appears at the junction of the fluids. This test is not to be relied on absolutely, as saliva and nasal mucus produce the same blue ring, and the urine of patients taking iodide of potassium gives a similar reaction.

The distinction between hæmaturia, or urine in which both the colouring matter and the corpuscles are present, and hæmoglobinuria, or urine which contains the colouring matter without the corpuscles, has already been drawn.

Hæmaturia may be caused by renal affections such as nephritis, pyelitis, and cancer; by inflammation or malignant disease of the urinary channels; and by morbid conditions of the blood, such as scorbutus and purpura, or the presence of the *Bilharzia hæmatobia*.

Hæmoglobinuria, in which the blood is disintegrated within the circulatory system, follows the absorption of certain toxic agents, such as chlorate of potassium, carbolic acid and pyrogallic acid; it may be the result of insolation, or it may be spontaneous as in the disease termed "paroxysmal hæmoglobinuria."

Bile gives the urine various shades of colour, from dark yellow to greenish brown. Its presence may be proved by tests for the bile acids and the bile pigments.

For *bile acids* ($C_{26}H_{43}N_6O$ and $C_{26}H_{45}NSO_7$) the best process is the modification of Pettenkofer's test suggested by Francis and described by Ralfe.[1] This method consists in the use of sulpho-saccharic acid. The reagent is made by drying 30 grains of glucose over a water bath, and dropping it when cold into half an ounce of strong sulphuric acid. If the glucose is quite dry it does not become charred, but forms, with the acid, a straw-coloured liquid which keeps for some days if closely stoppered and placed in the dark. A test-tube is filled to the height of half an inch with this reagent, and the urine is floated upon the surface. A beautiful purple colour at the line of junction is characteristic of bile acids.

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[1] *A Practical Treatise on Diseases of the Kidneys and Urinary Derangements*, by Charles Henry Ralfe, M.A., M.D.; London, 1885, page 113.

The *bile pigments* ($C_{32}H_{36}N_4O_6$ and $C_{32}H_{36}N_4O_8$) are recognised by the play of colours, in which green is the distinctive character, produced by the action of several reagents.

Gmelin's test is performed by placing a few drops of the urine to be examined on a white porcelain plate, and near it a few drops of fuming nitric acid. The two fluids are to be gently brought into contact, when the play of colours, and particularly the green tint, must be looked for.

Fleischl's test consists in mixing equal parts of the urine and nitric acid in a tube, and underlaying the mixture with sulphuric acid. The green tint and play of colours should be seen at the line of junction.

Heller's test consists in mixing equal bulks of urine and hydrochloric acid, the mixture being underlaid with nitric acid. The appearances in this test should be the same as in that last mentioned.

Marechal's test is performed by floating a small quantity of urine upon the surface of some tincture of iodine in a test-tube. A beautiful green colour will appear at the line of contact if bile pigment is present.

By means of the spectroscope no absorption bands of bile acids or pigments can be found unless some reagent has been added previously. If fuming nitric acid be added to urine containing bile, and this be examined with the spectroscope, two absorption bands will be seen, one being in the orange and yellow, the middle point of which is about the line D, another being in the green, extending from near C to a little beyond F. If the action of the acid on the bile is allowed to go on longer, it will be found that the latter band alone can be seen. It is identical with that of urobilin already described (page 248), and is produced by the oxidation of bilirubin into urobilin.

MacMunn[1] describes and figures the spectrum of Pettenkofer's test for bile acids as giving a narrow band between C and D and a broad band at E.

The presence of bile in the urine is significant of some interference with the passage of bile from the liver into the duodenum, the old views as to the occurrence of hæmatogenous jaundice being erroneous. In recent cases of jaundice the bile acids are found in considerable quantity, but in the urine of patients who have suffered for some time the amount of the acids is smaller, or they be altogether absent.

Glucose ($C_6H_{12}O_6$).—Healthy urine usually contains traces of glucose, but in so minute a quantity that it may be regarded as an abnormal substance. If the specific gravity of any urine is 1030 or more, it raises an expectation that glucose is present; and if the urine is at the same time pale in colour and excessive in quantity, the expectation is almost a certainty. It may be detected by means of several tests, all depending upon the fact that when glucose is boiled with caustic potash it is oxidised—taking oxygen from any source available, and hence causing reduction.

Moore's test consists in the mixture of equal parts of urine and liquor potassæ in a test-tube, the upper layer of which is to be boiled. If sugar is present, a ruddy brown colour is developed in the heated zone from the formation of glucic and mellisic acids. If nitric acid be now added, the brown colour is discharged, and an odour of caramel is given off.

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[1] *The Spectroscope in Medicine*, by Charles A. MacMunn, B.A., M.D.; London, 1880, page 165.

This is by no means a reliable test, and is apt to be vitiated by impurities in the solution of potash.

Trommer's test is performed with liquor potassæ and a weak solution of sulphate of copper, the strength of which should not be greater than 1 in 25. It is conducted by adding a drop or two of the copper solution to the urine, and then a volume of the solution of potash equal to that of the urine. On adding the potash solution a blue precipitate of hydrated cupric oxide is thrown down; and if sugar is present, this is again dissolved on shaking the tube, and a clear blue fluid results. This is characteristic of the presence of sugar. On boiling the mixture, if sugar is present, a dense yellow precipitate of hydrated cuprous oxide is produced through the reduction of the cupric oxide by the sugar, and this yellow precipitate is afterwards changed into a red deposit of cuprous oxide by the loss of water.

Proteids and uric acid compounds have a slight reducing power over cupric compounds, and their possible influence must always be remembered when the reduction is slight.

Fehling's test.—This is simply a modification of Trommer's test, in which the cupric oxide is held in solution by an alkali and an organic compound. The reagent employed is composed of—

Cupric sulphate, 34·64 grammes, dissolved in 500 cubic centimetres distilled water.

Neutral potassium tartrate, 173 grammes, dissolved in 500 cubic centimetres solution of caustic soda (specific gravity 1·12).

These should be kept in separate bottles, and mixed when required in equal proportions.

The strength of this solution is so adapted that 10 cubic centimetres are reduced by 0·05 gramme of glucose; and, as will be mentioned below, this renders the test of use in the quantitative estimation of sugar in the urine.

As tartaric acid is apt, if long kept, to be changed into racemic acid, which will reduce cupric to cuprous salts, the solution must be boiled by itself before being employed as a test.

The best manner of employing this test is as follows:—Fill a test-tube to the depth of an inch with Fehling's solution, boil it cautiously and set it aside to cool. If no deposit occurs the solution is in good order. Heat it again to the boiling-point, and add a drop of the suspected urine; if sugar is present, a yellow precipitate is suddenly formed, which turns red on longer boiling or standing aside. If the sugar is small in amount more urine must be added, but an excess of urine must be guarded against, as excess of sugar dissolves the copper salt.

Serum albumin and serum globulin must be remembered, if present, and all substances belonging to the uric acid group must be taken into account. Certain drugs taken internally give a reaction with Fehling's solution. Amongst these are ferrous sulphate, gallic and tannic acids, chloroform and resins.

Pavy's test.—The solution in this case is prepared by dissolving 4·158 grammes of cupric sulphate in 200 cubic centimetres of distilled water by heat; 20·4 grammes of potassium sodium tartrate, and the same quantity of caustic potash, in 400 cubic centimetres of distilled water; mix the two solutions gradually; when cold, add 300 cubic centimetres of strong ammonia (specific gravity ·88), and add enough water to make a litre.

This is to be used in the same way as Fehling's solution. It has the advantage of giving no precipitate, as the ammonia holds the reduced cuprous oxide in solution. Its disadvantage is the giving off of ammonia fumes. The complete reaction is indicated by the disappearance of the colour.

The same precautions must be taken as in the case of Fehling's solution, and the same fallacies may be found if these are not attended to.

Bötger's test is based on the fact that glucose has the power of reducing the salts of bismuth. It consists in mixing equal volumes of urine and liquor potassæ in a test-tube and adding a pinch of bismuth sub-nitrate. On boiling, if glucose is present, a deposit of metallic bismuth will be thrown down, which will be grey if the sugar is only present in small amount, and black if in larger quantity.

As albumin and globulin have the power of reducing bismuth salts also, they must be taken into account.

Johnson's test.—Although originally suggested by Braun, this process has only been commonly employed since its revival by Johnson, whose name it usually bears. The test consists in mixing a drachm of the suspected urine with half a drachm of liquor potassæ and forty minims of a saturated solution of picric acid. If albumin or globulin be present a precipitate is formed, but this does not cause any interference with the reaction. On boiling the upper layer a deep red colour is produced, if sugar is present, by the reduction of the picric to picramic acid.

The *fermentation test* is an exceedingly useful method not only of detecting the presence, but also of approximately estimating the amount, of glucose in urine. It is best conducted in the differential manner described by Roberts. Collect and measure the urine of twenty-four hours: place four ounces of it in an eight-ounce bottle, with a small piece of German yeast, and four ounces in a similar bottle without any yeast. Lay the bottles aside in a warm place (from 20° to 25° C.) for twenty-four hours. At the end of this period the specific gravity of each specimen is to be ascertained with the urinometer. If sugar is present, it will be found that the specimen to which the yeast was added has a lower specific gravity than the other. Roberts has proved by means of a careful series of experiments that the loss of specific gravity gives an index to the amount of sugar present. Each degree lost represents 0·22 gramme of glucose in 100 cubic centimetres of urine, or 1 grain per ounce. If the urine contains less than 0·5 per cent., or $2\frac{1}{2}$ grains per ounce, it gives no reaction with the fermentation test, because the fluid absorbs all the carbon dioxide which is given off during the process. Recent researches have shown this method to be very unreliable.

By *polarimetry* the presence of sugar may be detected with great readiness, and the quantity present can be estimated. The method is based on the fact that glucose has the power of rotating polarised light towards the right, and the amount of deviation is an index to the amount present. It is by no means such a delicate test for glucose as many of the chemical processes, but is an exceedingly rapid one.

There are several different kinds of instruments—the name of which is the *saccharometer*—all of which are constructed on similar lines. The light passes through one prism called the polarising, and afterwards through another called the analysing prism. If these be so placed that the oblique ends of the two prisms are parallel, the polarised part of the ray of light—that which is allowed to pass through the first prism, while the other part of the ray is absorbed—passes through the analyser without obstacle. If the analyser is turned on its axis the light will become more and more obscured until an angle of 90° has been reached, at which point it will be quite shut out. If it is rotated beyond this angle the light will gradually return until 180° is reached, when it is allowed to pass freely. It again diminishes to 270°, and increases to 360°.

If the prisms are so arranged that the light passes without obstruction, it will be found that a column of urine containing glucose causes partial obstruction by rotating the light towards the right, and that if the analysing prism is rotated, the light will pass when a certain point has been reached. The rotatory power of glucose is 57·6°. If the length of the column of the fluid containing glucose is known, and the angle of deviation is noted, the per-centage of sugar can be calculated.

If highly coloured, the urine must previously be freed from colouring matter by the addition of a solution of acetate of lead and subsequent filtration, or by clarifying it by passage through animal charcoal. If the solution of lead acetate has been employed, one-tenth must be added to the result.

If the length of the tube containing the urine is one decimetre, and the amount of deviation is 8·69, then—

$$100 : 57{\cdot}6 :: 8{\cdot}69 : 5$$

the urine contains 5 per cent. of glucose. It must be remembered, however, that all instruments are not graduated alike.

The *quantitative estimation* of glucose may be conducted most accurately by means of the titration process with Fehling's solution, of which, as has been already said, 1 cubic centimetre is equivalent to 0·05 of a gramme of sugar.

For this method a graduated burette and stand are required. The process is conducted in the following manner :—

Take 10 cubic centimetres of Fehling's solution, dilute it with 40 cubic centimetres of distilled water, and boil. Place 10 cubic centimetres of urine diluted ten times with water in the burette, and from this drop half a centimetre into the hot Fehling's solution. A yellow or red precipitate will fall at once to the bottom. After it has subsided, add another half centimetre, and so on until all the blue colour has disappeared. The exact moment of its disappearance must be noted. If 10 cubic centimetres of urine are required to decolorise all the diluted Fehling's solution, which, as above mentioned, is equivalent to ·5 gramme of glucose, the 10 cubic centimetres contain exactly this amount. From this it is easy to calculate the per-centage as follows :—

$$10 : 100 :: {\cdot}5 : 5$$

the urine will contain 5 per cent. of glucose.

The *clinical significance* of sugar in the urine is extremely wide, but there are two great classes of cases in which it appears. It may be present as a temporary constituent, caused by a surplus of carbo-hydrates in the system or by some transient disturbance of the hepatic vaso-motor apparatus; or it may be permanently present in cases of profound disturbance of the glycogenic function.

In certain conditions the peculiar odour of *acetone* (C_3H_6O) may be detected in the urine. This substance may be recognised by several tests, but that which is most available is the nitro-prusside of sodium reaction. On the addition of a solution (·1 gramme to 15 cubic centimetres of water) of nitro-prusside of sodium, along with caustic potash or caustic soda, to urine containing acetone, a ruby red tint is produced, which slowly fades into a straw colour. The same colour is produced by these reagents in the presence of ethyl-diacetic acid ($C_6H_{10}O_3$), aldehyde, and kreatinin. In the case of the two last mentioned, however, the ruby red rapidly becomes a straw yellow, and on the subsequent addition of an acid no change occurs; while with acetone the addition of an acid produces a violet colour, and with ethyl-diacetic acid it gives a dark tint. If the solution of sodium nitro-prusside be employed along with nitric acid, a rose violet colour is slowly given by acetone, and a ruby red, slowly fading into straw yellow, by ethyl-diacetic acid; no change occurs with aldehyde or kreatinin.

The clinical significance of acetone is as yet somewhat obscure. Formerly it was supposed to occur only in connection with grave forms of diabetes, and more especially with the development of the fatal complication known as Küssmaul's coma. That its occurrence is much more general is now known. According to Saundby it has been detected—

1*st.* In many acute diseases, *e.g.*, pneumonia, without symptoms of coma being present.

2*d.* In cancer, Bright's disease, perityphlitis, strangulated hernia, after minor surgical operations, and sulphuric acid poisoning, without dyspnœa or coma.

3*d.* In a case of cancer of the stomach dying from coma (von Jaksch). Acetone and diacetic acid were present, but no sugar, in the urine of this case.

4th. In a case of Litten's after scarlatina, in which albuminuria was present.

5th. In diabetic urine, during or just before the peculiar terminal dyspnœa sets in.

It may be added that in the case of Cetti, who starved in Berlin for fourteen days, acetone was largely present in the urine, and Senator considers its presence an inanition symptom indicative of grave disturbance of the metabolism.

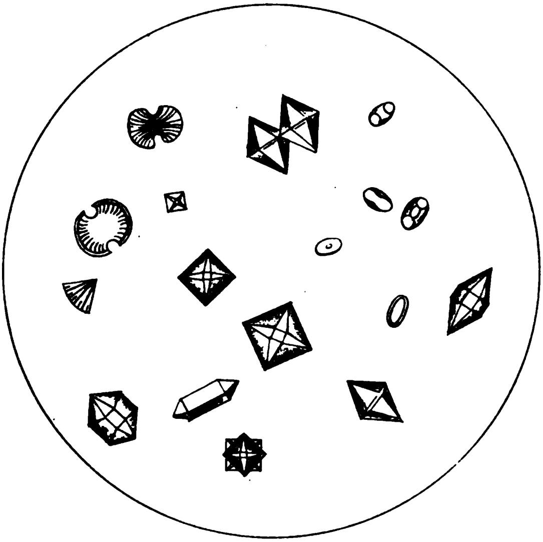

FIG. 81.—Calcium oxalate.

Along with acetone must be considered *ethyl-diacetic acid* ($C_6H_{10}O_3$).

This substance readily takes up water, and splits into acetone and alcohol—

$$C_6H_{10}O_3 + H_2O = C_3H_6O + C_2H_6O + CO_2.$$

With a solution of ferric chloride it strikes a deep red colour, and since this reaction, although not given either by acetone or alcohol, is exceedingly common, while acetone is manifest by its odour, it has been supposed that this diacetic acid is the source of the acetone. The exact relationship of these two bodies to one another and to Küssmaul's coma is still far from clear. Recently, the discovery in the urine of diabetes of a substance closely allied to β-hydro-oxybutyric acid, from which apparently diacetic acid is readily produced, has further complicated the question.

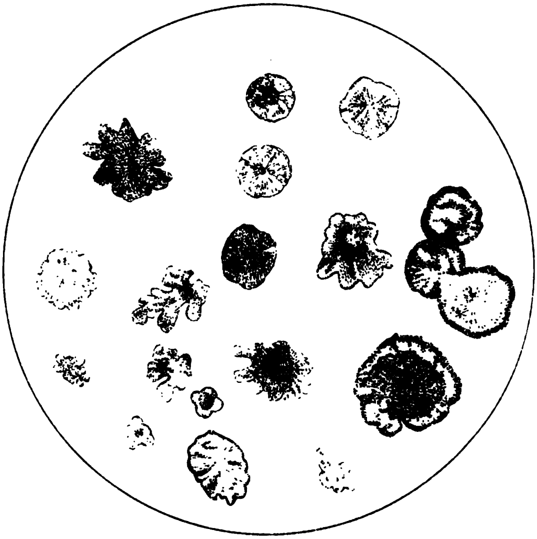

FIG. 82.—Leucin.

Certain *crystalline substances* require to be noticed amongst the abnormal constituents of urine.

Oxalate of Calcium is deposited in the urine of patients suffering from various disturbances of the metabolic processes. It assumes the form of octahedral crystals and masses resembling dumb-bells, as seen in Fig. 81.

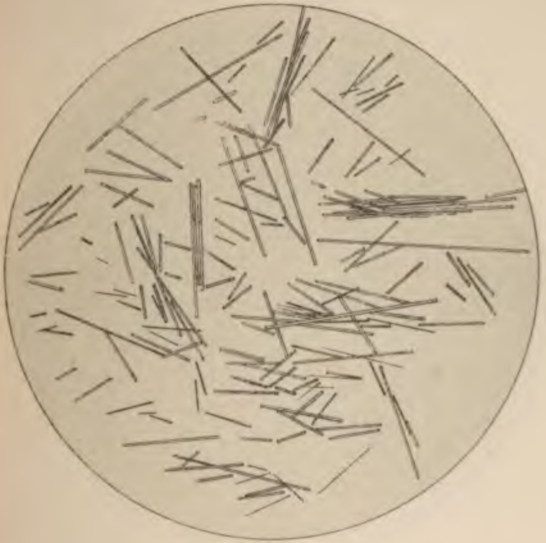

FIG. 83.—Tyrosin.

Leucin and tyrosin, when present in the urine, are always found together. If present in the urine in considerable proportion, the evaporation of a drop is sufficient to give evidence of their presence, but they are usually present in such small amount as to require separation by artificial means.

Leucin may be obtained by evaporating the urine to dryness and dissolving the residue in boiling alcohol. When cold the solution will deposit leucin in the form of a greasy mass. Examined under the microscope, leucin is found to be composed of spherical bodies, with concentric markings interrupting a radiated structure.

Tyrosin may be obtained from the urine by precipitating the colouring matters with basic acetate of lead, filtering the mixture, and decomposing the filtrate by means of sulphuretted hydrogen. On evaporation tyrosin separates out. It presents the appearance of long acicular prisms, sometimes separate, at other times combined as sheaves and spherical bodies.

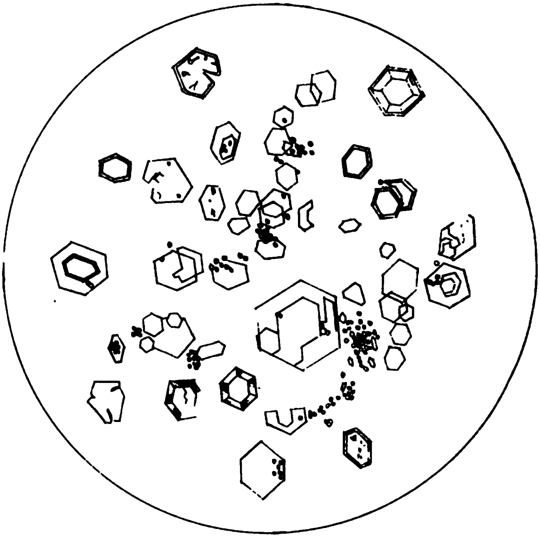

FIG. 84.—Cystin.

The *clinical significance* of leucin and tyrosin is very precise. Their presence is characteristic of destruction of the hepatic cells, and they are most commonly to be found in the rapid disintegration of the liver, of acute yellow atrophy, phosphorus poisoning, and some malignant forms of fever, as well as various hepatic disorders.

Cystin is an uncommon urinary deposit, and forms a rare constituent of urinary calculi. If it is present in urine it may be separated by adding acetic acid in excess and allowing the urine to stand for a day. The precipitate is to be collected on a filter, dried, and dissolved in ammonia, from which cystin, if present, will be deposited on evaporation.

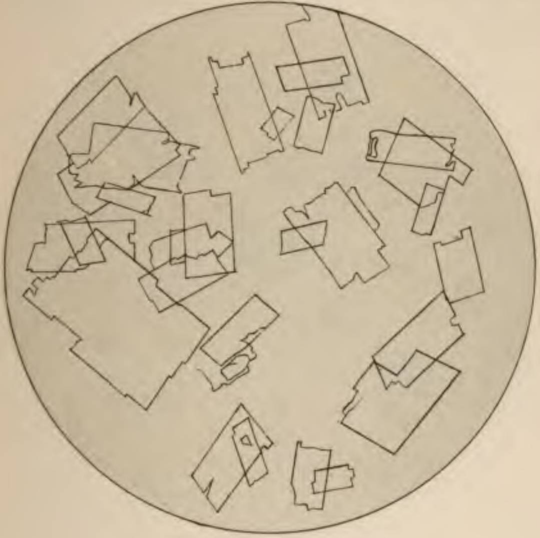

FIG. 85.—Cholesterin.

Under the microscope cystin appears usually in the form of hexagonal crystals superimposed upon each other, but it is occasionally seen as rhombohedral prisms, scattered or in groups.

Cystin may be detected by boiling urine containing it with a solution of acetate of lead in presence of caustic potash. It contains sulphur, which on decomposition gives a dark precipitate of lead sulphide.

The *clinical significance* of cystin is rather obscure. It occurs in some hepatic disorders, and has been found in tubercular and anæmic conditions.

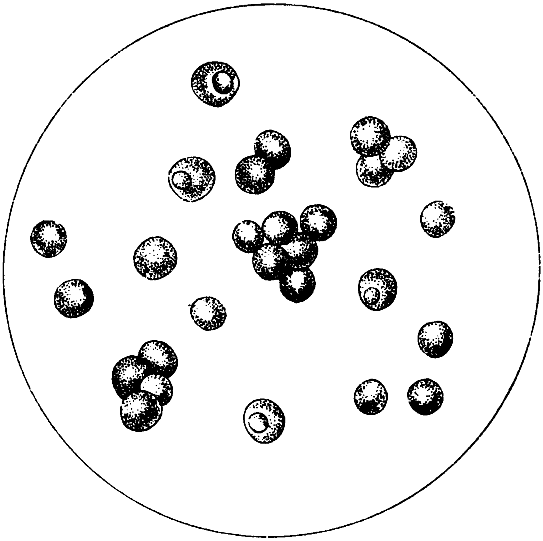

FIG. 86.—Mucus and pus.

Fatty matters are normally present in the urine in traces, one grain being obtained from about two quarts of urine. In chyluria, as already mentioned, fat is present in large proportion, and this has been determined to consist of neutral fats, cholesterin, and lecithin.

Cholesterin is a monatomic alcohol sometimes deposited from urine, occurring in the form of rhombic plates with notches at their angles.

Its *clinical significance* is not very clear, but it is usually regarded as the result of the absorption of purulent matter.

Substances derived from the genito-urinary tracts.—Several of these substances are normally present, such as epithelial and mucus cells; others are abnormal constituents of urine, as pus cells and tube-casts. It is well, however, to consider them together—in fact it would be highly inconvenient to separate them.

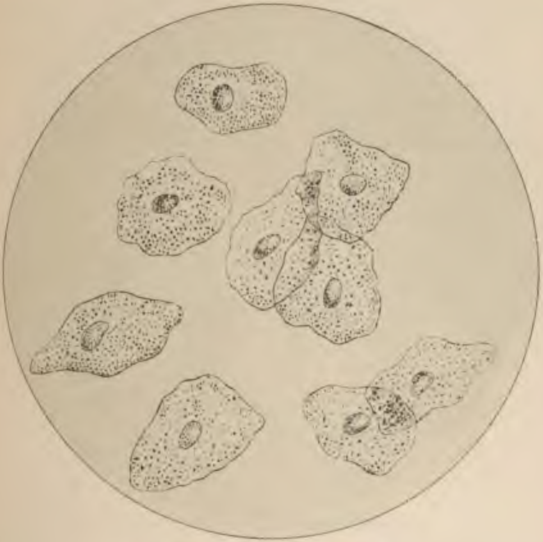

FIG. 87.—Squamous epithelium.

Mucus.—The light transparent cloud of mucus, previously described, consists of mucus corpuscles and nucleated epithelial cells. It varies much in quantity, and under abnormal conditions may have the addition of other substances, such as pus and blood.

If acetic acid is added to mucus it produces a deposit of stringy mucin, and the same appearance may be found in highly acid urine. Mucin is soluble in caustic alkalies and in solutions of lime and baryta. It is not precipitated by heat or by mercuric chloride—a fact which serves to distinguish it from albumin and globulin. Dilute mineral acids cause a precipitate soluble in excess. Alcohol, acetic, citric, and picric acids coagulate it.

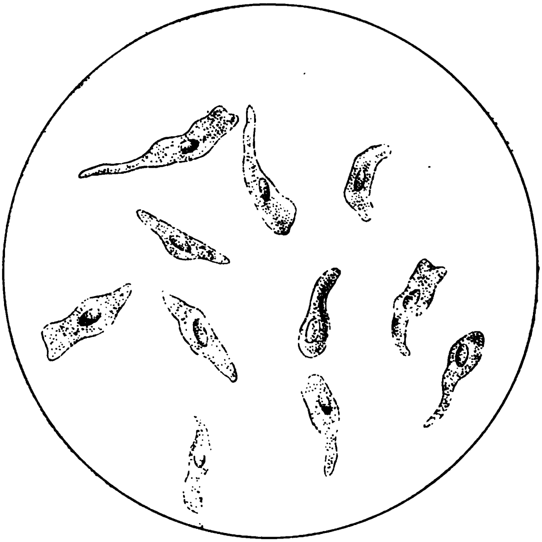

FIG. 88.—Columnar epithelium.

The *epithelial cells* found in the mucus cloud present different characters according to their origin.

Mucus cells are small round or oval bodies, about the size of a white blood corpuscle. They swell up on the addition of water or dilute acetic acid (Fig. 86), which render the nuclei distinct.

Squamous epithelial cells (Fig. 87) are derived from the bladder or vagina. They are large in size and irregularly circular in outline, presenting distinct nuclei. Cells from the vagina are larger than those thrown off by the bladder, but it is often very difficult to distinguish the one from the other.

Columnar epithelial cells have their origin in the urethra, ureter, or the pelvis of the kidney. They are irregular in shape, presenting cylindrical, pyramidal, or fusiform outlines. Their form gives no indication of their origin, which can only be ascertained by the accompanying symptoms (Fig. 88).

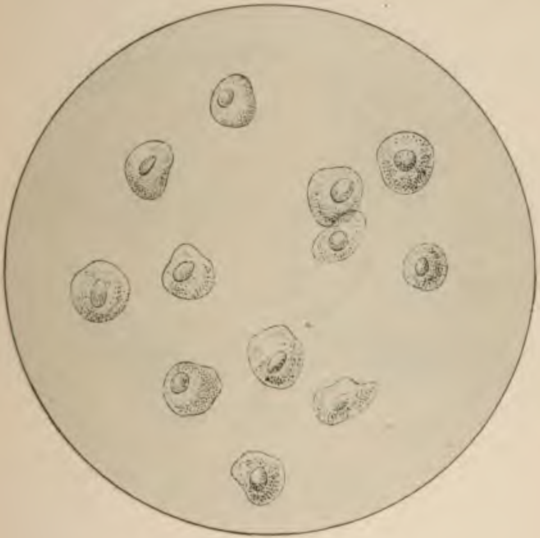

FIG. 89.—Round epithelium.

Round epithelial cells with distinct nuclei are of renal origin, and result from some change in the tubules of the kidney (Fig. 89). They will be referred to again, in dealing with tube-casts.

Pus.—When pus is present in acid or neutral urines it forms an opaque white deposit, but in alkaline urines it occurs as a gelatinous ropy precipitate. Pyin is coagulated by solutions of mercuric chloride, which distinguishes pus from mucus: the fact that liquor potassæ forms with pus a viscid ropy substance like white of egg is a useful test for it.

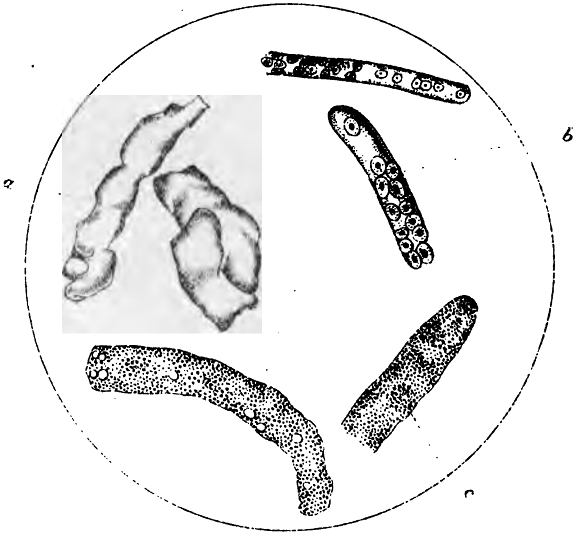

FIG. 90.—Amyloid (*a*), epithelial (*b*), and granular (*c*) tube-casts.

Under the microscope pus cells cannot be distinguished from mucus cells.

It is impossible to distinguish the albumin of the liquor puris from albumin transuding from the kidney.

Tube-casts.—Tube-casts are the result of renal inflammations, and are produced, as Ralfe well describes it, by an albuminous exudation into the tubules, forming moulds or casts. If these are washed away without the separation of epithelial cells, *hyaline casts* are found; if blood corpuscles are mingled with them *blood casts* are observed; if epithelial cells adhere to them *epithelial casts* are seen; if the epithelial cells have undergone degeneration *granular* and *fatty* casts make their appearance. Sometimes the hyaline casts undergo *amyloid* degeneration. They are then termed *waxy* casts, and give a deep stain with iodine and methyl-violet.

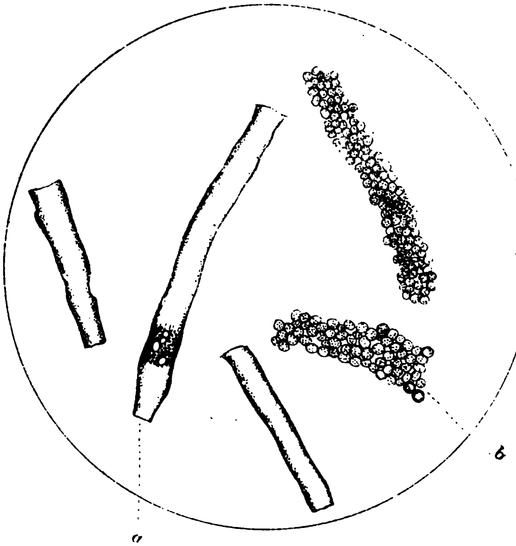

FIG. 91.—Hyaline (*a*) and blood (*b*) tube-casts.

The *clinical significance* of tube-casts is exact. Their presence is indicative of inflammation of the kidney. Recent cases of nephritis give rise to hyaline, blood, and epithelial casts, while cases of older standing are accompanied by granular, fatty, and waxy casts.

CHAPTER IX.

THE NERVOUS SYSTEM.

FOR obvious reasons, the investigation of the nervous system is concerned more with the observation of functions than with the examination of organs, and, as a consequence, the diagnosis of nervous disorders is beset with much greater difficulty than is the case with any other group of diseases. A thorough grasp of the subject can only be attained by acquaintance with the facts of medical anatomy and physiology, and their application to the symptoms in any given case. The consideration of the nervous system therefore falls naturally into an introductory sketch relating to anatomical and physiological facts, and an investigation into the symptoms presented in disease.

MEDICAL ANATOMY AND PHYSIOLOGY OF THE NERVOUS SYSTEM.

In this section only such facts as are of real use in the investigation of disease will be mentioned, and, as the greatest practical assistance in diagnosis lies in a correct knowledge of the paths of nervous impulses, the introductory observations which follow will in the main be devoted to tracing the lines along which such nervous impulses are transmitted.

1. *Paths of conduction of impulses of ordinary sensation.*—From their terminations in the different sensory end-organs, the fibres concerned in the transmission of impulses from the periphery to the centres run in the mixed nerves until they enter the spinal cord by the posterior roots. On arriving in the cord some of the fibres pass directly into the grey matter

of the posterior horn, and others proceed along the external column for a short distance. These latter seem afterwards in part to enter the posterior horn, and in part to continue their course in the external column. The exact course which they take after this point is still a matter of doubt, but from pathological observations, and for clinical purposes, it may safely be assumed that a total decussation of the fibres takes place shortly after their entrance into the cord, so that, with the exception of a few fibrils which establish connections with different parts of the grey matter of their own side, all the fibres corresponding to one side of the body pass over to and proceed upwards in the opposite side of the cord. Their relative position in the cord is still a matter of discussion. Certain authorities believe that their upward course lies in the posterior columns, while others are of opinion that they pass up in the lateral columns of the cord. The weight of evidence is in favour of the latter view. The paths of sensory conduction seem to pass upwards, at least in part, by means of the restiform bodies, through the pons, along the crus and internal capsule, and appear finally to end in the temporo-sphenoidal and hippocampal region, which are probably the regions wherein the centres of ordinary sensibility are localised. The following diagram gives the course of the paths of sensory conduction (Fig. 92).

The paths by which impulses of deeper sensation—usually known as the "muscular sense"—are conducted seem to be similar to those just described, but their terminations are at present quite unknown.

2. *Paths of conduction of impulses of special sensation.*—It must be borne in view that of the sensory cranial nerves the olfactory and optic pass directly into the substance of the brain, while the sensory fibres of the trigeminal, auditory, glosso-pharyngeal, and pneumo-gastric nerves enter by means of the middle oblongata.

The *olfactory path* appears to be through the roots of the olfactory nerves into the surface of the brain, where they

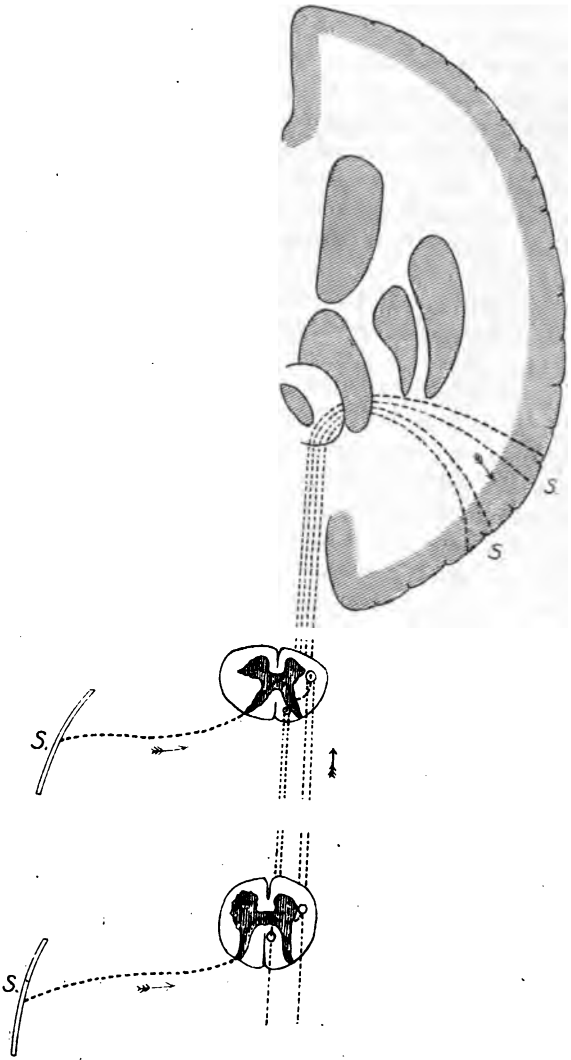

FIG. 92.—Paths of conduction of sensory impulses.

probably decussate, pass along the internal capsule, and end in the temporo-sphenoidal region.

The *visual path* is by means of the optic nerves (which undergo a special and peculiar decussation in the optic chiasma, to be afterwards more fully referred to), along the optic tracts to the corpora geniculata, corpora quadrigemina, and optic thalamus, whence some of the fibres pass through the internal capsule, and reach the cortical centre of vision, situated in the occipital lobe and perhaps also in the angular gyrus.

The *auditory path* has its course along the auditory nerve to the nuclei in the floor of the fourth ventricle, where decussation probably takes place, and whence some fibres pass to the cerebellum, and others apparently pass up through the internal capsule, to end in the temporo-sphenoidal region.

The *gustatory path* is much more complicated. The posterior part of the tongue is brought into relationship with the centres by means of the glosso-pharyngeal nerve directly. This enters the medulla, and passes to a nucleus in the floor of the fourth ventricle. Some of the fibres appear to pass up the internal capsule, and to reach the inner and upper part of the temporo-sphenoidal region. The anterior part of the

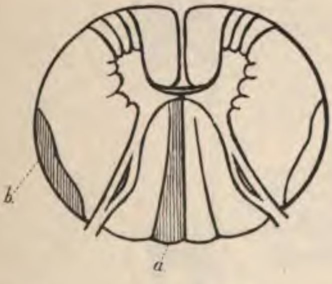

FIG. 93.—The postero-median columns, or columns of Goll (*a*), and the direct cerebellar tracts (*b*).

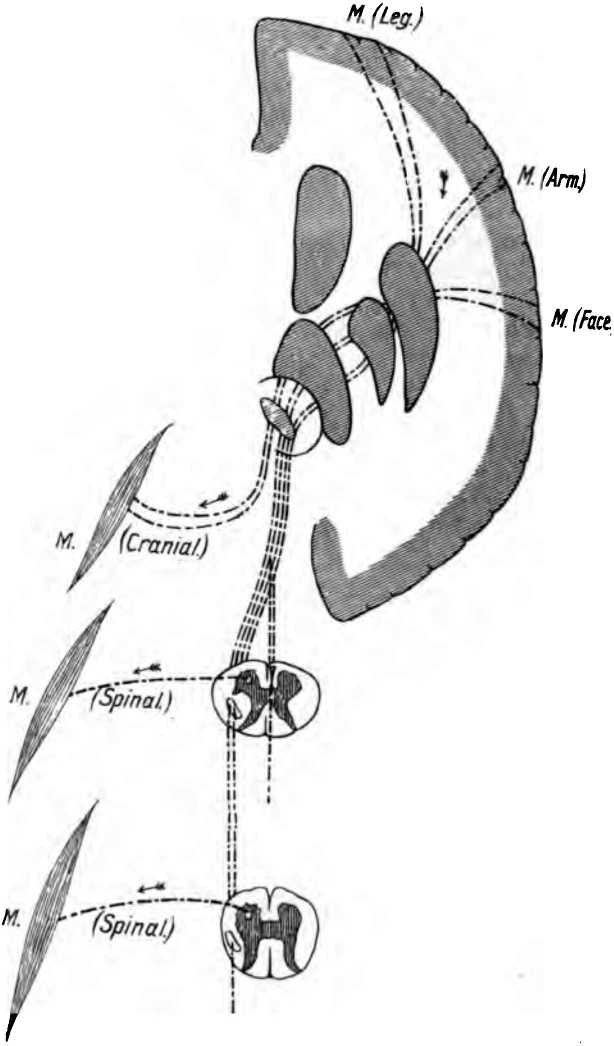

Fig. 94.—Paths of conduction of motor impulses.

tongue seems to be related to the centres by the chorda tympani, connected first with the lingual and afterwards with the facial nerve, whence its course lies through the great superficial petrosal to the spheno-palatine ganglion, from which it passes to the trigeminal, and accompanies it to the medulla.

Although hardly to be termed sensory paths, there are certain tracts which must be mentioned here, as they certainly transmit impulses upwards. The principal paths of this kind are the postero-median columns, and the direct cerebellar tracts of the spinal cord (Fig. 93). Nothing is known with certainty in regard to the functions of these structures, but it may be observed that they are the seat of the structural changes known as "ascending degeneration."

3. *Paths of conduction of motor impulses.*—The motor paths have their origin in the motor areas, situated in the cerebral cortex around the fissure of Rolando. For an exact description of these areas special works must be consulted, but it may be mentioned that the area for the leg is situated near the vertex, that for the arm lower down, and that for the head and face at a still lower level. The motor paths converge towards the internal capsule through which they pass, and proceed by means of the crura cerebri to the pons and medulla. In the medulla most of the fibres decussate,

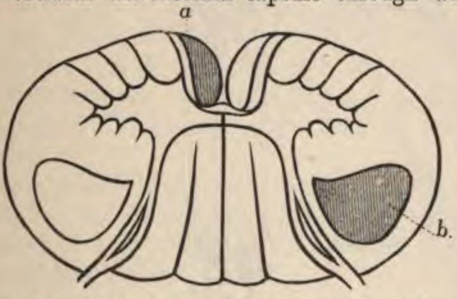

FIG. 95.—Direct (*a*) and crossed (*b*) pyramidal tracts, in which are the motor paths corresponding to the left side of the brain.

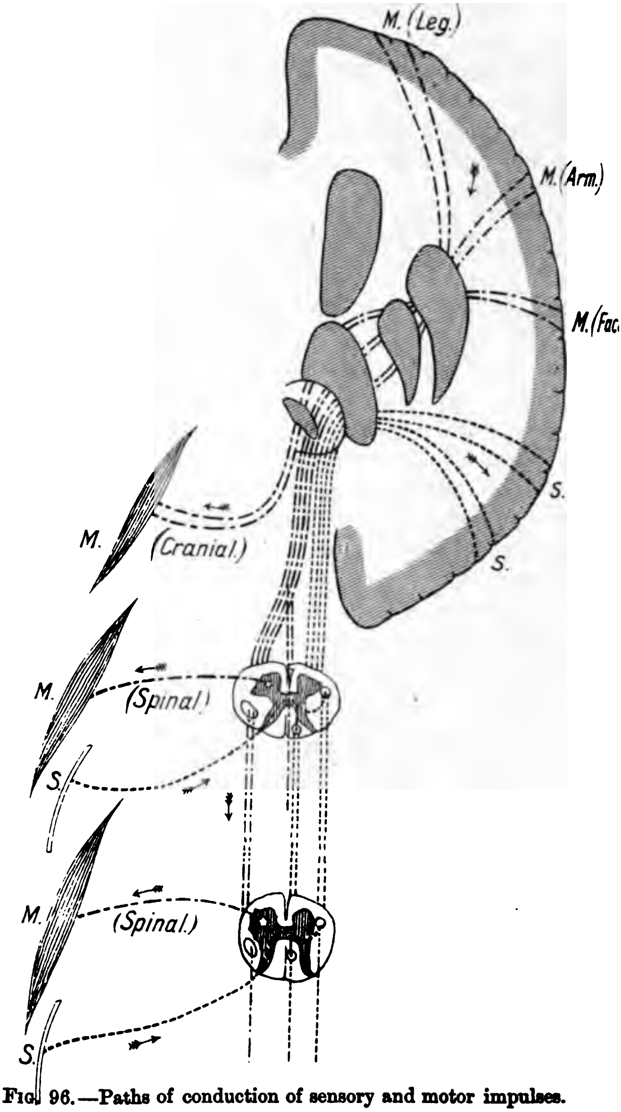

FIG. 96.—Paths of conduction of sensory and motor impulses.

forming the *crossed pyramidal tracts*, but a certain number pass downwards for some distance on the same side as that from which the paths originally rose, and form the *direct pyramidal tracts.*

The terminations of these fibres are somewhat various. Some appear to pass into the grey matter and end in its cells ; others seem to pass into it and emerge again as part of the anterior nerve-roots.

The fibres concerned in conducting motor impulses pass out in several bundles, forming the anterior nerve-roots, from which the various mixed and motor nerves have their origin, and finally reach the muscles, where they terminate in the motor nerve endings. The diagram on page 290 gives a connected view of the relationship of these paths (Fig. 94).

The cranial motor nerves appear to arise in different parts of the brain, but most of their cortical origins have not yet been discovered. It is of importance to remember that the oculo-motor and trochlear nerves have an origin from a nucleus below the aqueduct of Sylvius, and that they emerge from the brain respectively on the inner and outer aspects of the crus cerebri. It is of equal importance to remember that, whatever may be their cortical connections, the motor fibres of the trigeminal, abducent, facial, glosso-pharyngeal, pneumo-gastric, spinal accessory, and hypo-glossal nerves have intimate relations with nuclei in the floor of the fourth ventricle, and that they emerge either from the pons or from the medulla.

The main lines along which motor impulses are transmitted in the spinal cord are, as already mentioned, the direct and crossed pyramidal tracts, which are to be seen in the accompanying diagram (Fig. 95).

The diagram (Fig. 96) on the foregoing page gives a view of both sensory and motor paths.

In closing these introductory remarks on the anatomical and physiological relations of the nervous system, stress should be laid on the facts that the brain and cord are at once centres and conductors, and that the cause of disorder of function in any region must be sought in some zone through which the impulses travel to or from the affected region.

THE INVESTIGATION OF NERVOUS SYMPTOMS.

We have now to consider the various symptoms presented by disease of the nervous system. This section naturally falls into a consideration of—

1. Sensory functions.
2. Motor functions.
3. Reflex functions.
4. Visceral functions.
5. Vaso-motor functions.
6. Trophic functions.
7. Mental functions.

These will now be discussed in the above order.

1.—SENSORY FUNCTIONS.

As this work is mainly devoted to *objective*, it is not possible to devote much space to *subjective* phenomena. It is, however, necessary to make a few brief remarks on some of the more common and important subjective symptoms. They are all the effects upon consciousness of some sensory impression, and may have their origin in any part of the sensory path between the end-organ and the centre, but the sensation experienced is always referred to the peripheral region of the sensory tract.

ORDINARY SENSIBILITY.

Among the subjective phenomena belonging to the nerves of ordinary sensibility which are of most importance are sensations of pain, tightness, weight, sinking, heat, cold, numbness, itching, creeping, tingling, pricking, and throbbing. These subjective symptoms vary greatly in degree, and several different kinds are often found associated together.

More complicated sensory symptoms may be found, the most remarkable being the sensation of giddiness, or vertigo, produced by a great variety of affections.

Of much greater importance than these subjective symptoms are the results obtained by testing the *sensibility* of the sensory nerves. The sensibility to different kinds of impressions should be tested by appropriate means, such as will now be mentioned, applied to the different regions on both sides of the body for the purpose of comparing them.

Sensibility to *touch* may be roughly gauged by laying a finger upon the patient while the eyes are closed, but for greater exactitude it is necessary to employ such an instrument as the æsthesimeter, by means of which it can be determined whether the patient is able to estimate the distance between the two points of contact with the skin.

Sensibility to *locality* may be ascertained by touching the patient while the eyes are shut, and asking him to point to the spot.

Sensibility to *weight* may be tested by laying objects of similar appearance and size, but of different weight, upon the part under investigation. Balls of leather containing different weights are commonly employed for the purpose.

Sensibility to *temperature* may be determined by applying cold and warm test-tubes, or cold and hot sponges, to the surface of the body.

Sensibility to *electricity* may be ascertained by employing a gentle faradic current in order to compare the healthy and affected areas.

Sensibility to *pain* may be estimated by pricking or pinching the patient.

Deviations from the normal condition exist as *hyperæsthesia* or increase, and *anæsthesia* or decrease, of sensibility. There are many forms of perverted sensibility, as, for

The simple or direct visual sensations are those of *light*, *colour*, and outline or plane *form*.

Combinations of these with motor impulses sent to the ocular muscles, aided by psychical processes of memory and comparison, give us conceptions of size, distance, and solid form. These are visual judgments.

To determine the functional power of the optic nerves and visual paths, it is sufficient to direct our attention to the state of the simple sensations.

In health and under suitable stimulation these may be called forth at any part of the retinal surface, except the optic disc (" the blind spot ") and near the ciliary processes.

The retinal sensibility, under ordinary illumination, is by no means uniform throughout. It is most acute at the fovea centralis, where the cones are most abundant, and fades away towards the periphery of the fundus.

Direct vision.—If we wish to see an object distinctly, we direct our eye towards it in such a way that its image falls upon the fovea centralis. We call this act *direct vision.*

But rays of light from a considerable area around the object looked at enter the eye through the pupil at the same time, and stimulate sensitive retinal elements.

Field of Vision for Light.—This area is termed the *field of vision* for light. The point on which our gaze is fixed is the *point of fixation*, and any objects visible in the field not occupying the point of fixation are said to be seen by *indirect vision*, their images being applied to parts of the retina which are eccentric or peripheral in relation to the fovea centralis.

A complete subjective examination should therefore determine the sensibility to light, colour, and form at every part of the fundus of the eye which is normally sensitive. In the great majority of cases, however, there is no necessity for such exhaustive inquiry, and for clinical purposes it will be found convenient to conduct the examination as follows, taking care *to test each eye separately*, first in direct and then in indirect vision.

I. The direct visual acuity for form should be tested for *distant* objects, first *without* and then *with* the aid of suitable glasses, if necessary.

It has been found experimentally that, provided an object is sufficiently well illuminated, and stands in sufficient contrast to its surroundings, it will be clearly seen if its retinal image subtends an angle of one minute at the nodal point of the eye (see note, page 314).

No one object can fulfil this condition if placed at different distances from the eye, since the farther it is removed the smaller becomes its retinal image.

Snellen's Test Type.—Starting from this basis, Snellen has prepared a series of black test types in horizontal rows on a white surface, of such dimensions that when the card is placed at 6 metres (about 20 feet) from a normal eye, each of the smallest letters subtends an angle of 5 minutes within the eye, and the breadth of each of its component limbs an angle of 1 minute.

The letters in the next row are uniformly larger, so that each would subtend the same angles as above, though removed to a distance of 9 metres (about 30 feet).

Similarly the other rows contain letters which when seen at 12, 18, 24, 36, and 60 metres respectively would subtend the same angles. The letters have otherwise no special relation to one another, they do not combine to form words, and thus do not easily become familiar.

The individual to be tested should stand with his back to a bright window, the types being hung on a level with his head on the opposite wall *at 6 metres distance.*

In a badly lighted room daylight may be excluded and artificial light employed. In this case the light should be placed near the test types, and shaded on the side nearest the observer. A steady lamp or gas flame should be used, and a note taken of its approximate brilliance, so that, if required, the examination may be subsequently repeated under conditions as nearly similar as possible.

Having covered one eye lightly with the palm of the hand —or, preferably, a disc of some opaque material, as cardboard or dull glass, which does not prevent free evaporation from the excluded eye— the patient is requested to read the letters aloud, commencing with the largest.

How to express visual acuity.—If at this distance he reads the smallest of the series successfully, his sense of form (visual acuity) *at the macula* is normal, and may be conveniently expressed as a fraction thus—V (vision) $= \frac{6}{6}$ or unity; the numerator expressing the distance at which he is standing, the denominator the distance at which the type should be seen by a normal eye.

Suppose that at this distance (6 metres) he is unable to read letters smaller than those which should be seen by a normal eye at 12 metres distance, his $V = \frac{6}{12}$, *i.e.*, only half of the normal, and the visual acuity may be similarly expressed throughout the whole scale from $\frac{6}{9}$ to $\frac{6}{60}$, the denominator in each case corresponding to the row of letters at which distinct vision ceases.

Some test cards for distance bear a row of smaller letters which should be read at 5 metres by a normal eye. Many persons having keen vision can distinguish these at 6 metres.

In such a case $V = \frac{6}{5}$, the fraction showing that the acuity is above the average. If the size of a room necessitates the application of the test at a less distance than 6 metres, the actual distance employed must be stated in the numerator, and proportionately smaller test type must be used to denominate normal vision.

Thus at 5 metres if $V = \frac{5}{5}$ it is normal, but it may be defective, as $\frac{5}{6}$, or $\frac{5}{18}$, or $\frac{5}{60}$. If at 4 metres $V = \frac{4}{4}$, it is probably normal, but strictly speaking the full distance of 6 metres should always be employed by preference, for reasons which we must now consider.

The Refraction of the Eye.—Thus far we have said nothing of the influence of the refraction of the eye on the visual acuity. It is evident that if there is some error in the refractive media, the patient's optic nerve may be quite healthy, and yet his vision will appear defective because an indistinct image is received on the rods and cones.

The advantage of testing a patient at 6 metres is that rays coming from any point at that distance are practically parallel when they enter the pupil, and thus afford a convenient standard for estimating the ocular refraction.

Emmetropia.—An *emmetropic* (*i.e.*, normal) eye is constructed so as to bring parallel rays to a focus on the retina without effort on the patient's part.

Ametropia.—If the eye be *ametropic*, these rays will not be thus passively focussed. The defect may arise from hypermetropia, myopia, or astigmatism.

Hypermetropia.—In *hypermetropia* (badly termed "long sight") the refractive media at rest are incapable of bending the rays sufficiently, so that they impinge on the retina before they have come to foci. To get a sharper image the patient has to use his accommodation, *i.e.*, increase the refractive power of his lens by the action of his ciliary muscle, or requires the aid of such convex spherical glasses as will cause parallel rays to become sufficiently convergent before entering the eye that the defective refractive power of the natural media is compensated for.

Myopia.—In *myopia* ("short sight") the refractive media are proportionately too strong, so that parallel rays are brought to foci before they impinge on the retina. Rays from a less distance, being divergent, will have their foci thrown farther back, and at a certain distance from the eye, varying in different individuals with the degree of myopia, will be accurately focussed on the rods and cones. Hence for clear vision a myope requires to get nearer an object than an emmetrope, or has to employ concave spherical glasses, which cause rays passing through them to diverge as if coming from a nearer point.

Astigmatism.—By *astigmatism* we mean that the rays from any one point are not equally focussed within the eye.

Simple hypermetropic or *simple myopic astigmatism.*—Thus in one meridian the refractive power of the media may be emmetropic, but in another, and usually that one at right angles to the first, it may be hypermetropic or myopic.

Compound Hypermetropic Astigmatism.—Sometimes hypermetropia is present in every meridian, but most marked in one, and least in that at right angles to it.

Compound Myopic Astigmatism.—Similarly with myopia. In astigmatism the patient can only obtain clear vision with the aid of cylindrical lenses.

Irregular Astigmatism.—Sometimes the refraction varies so greatly from meridian to meridian that it cannot be definitely estimated, nor can the vision be satisfactorily improved by spectacles.

In any case, then, in which *V* appears defective we must ascertain the state of the refraction, with a view of determining whether the failure is optical or nervous.

Subjective Examination of the Refraction.—In proceeding to make this examination much time may be saved by a few judicious questions as to the nature of the patient's difficulty in seeing. Thus one who asserts that he sees well at a distance but less well near at hand, and especially in artificial light, who complains of letters running together, of painful sense of straining of the eyes, or headache after continued near work, is probably *hypermetropic* if young, or *presbyopic*, or both, if over forty-five years of age.

One who sees badly at a distance, but reads the smallest print if held near, is generally *myopic*, and the nearness required is a fairly accurate measure of his myopia.

Regular Astigmatism may be suspected if there is a complaint of seeing imperfectly both far and near, and especially if lines which run in one direction are more evident than those at right angles to the former.

Irregular Astigmatism, if in addition the lines and objects appear distorted.

Having ascertained the last row of letters which the patient can see with either eye separately, keep one covered and place before the other a weak *convex* glass, such as +0·5 D Sph. (see Objective Examination, p. 347).

If *V* was previously $\frac{6}{6}$ with the naked eye, but is made less distinct with this lens, the refraction is practically *emmetropic*, and need not concern us further.

If this lens causes no diminution of the visual power, or actually improves V, replace it by a slightly stronger convex glass. Enquire again what type can be read, and so long as *V* is improved, gradually increase the strength of the lens.

Suppose that with the naked eye a patient has $V = \frac{6}{6}$, and that when +2 D Sph. is placed before it he has still $\frac{6}{6}$ (the letters appearing larger and better defined), we conclude immediately that he is *hypermetropic*, that he obtained the first result by exerting his accommodation, and the second by relaxing it when the artificial lens gave the necessary degree of convergence to the rays before they entered his eye, and made an alteration of the curvature of his own lens unnecessary.

All the hypermetropia which we can reveal in this way is called "*manifest hypermetropia.*" If the patient is young it is probable that he has some more hypermetropia "*latent,*" that is, concealed and corrected by an habitual accommodative action of his ciliary muscle, which he is incapable of altogether relaxing. By the use of a mydriatic which paralyses the ciliary muscle the latent hypermetropia may be made manifest. It will be found that a stronger convex glass (say +6 D) is now needed to give the most distinct distant vision, and this lens is the measure of the "*total*" hypermetropia. In practice it is usually sufficient to order glasses which correct the manifest hypermetropia, unless there be convergent strabismus which it is desired to counteract by relieving the associated accommodative action.

In adults of forty years old and upwards, owing to the tendency to failure of accommodative power, all the hypermetropia, is usually *manifest*, and there is no need, and possibly some risk, in using a mydriatic, since it may cause an increase of the intraocular tension. In such a person the examination may show $V = \frac{6}{18}$ with the naked eye, but with +4 D Sph. $= \frac{6}{6}$. Here it is evident that the refractive media, even aided by such accommodative power as remains, are unable to bring parallel rays to a focus upon the retina without the above artificial assistance, and we declare that the patient has four dioptres of manifest hypermetropia.

By commencing this subjective enquiry with *convex spherical* glasses, the best inducement is given to the patient to relax his ciliary muscle. If concave glasses be first presented to a hypermetrope they are apt to excite, especially in the young, a strong ciliary contraction to overcome their dispersive action, which, if it does not readily relax, may completely prevent the true state of the refraction being determined by this method of enquiry.

If V be improved by convex spherical glasses, but be less than $\frac{6}{6}$, the convex spherical glass which gives the best vision should be retained in front of the eye, and a weak *convex cylindrical* lens added to it, and rotated slowly through two right angles, to determine in what axis, if any, its assistance is required. If benefit results, the case is one of *compound hypermetropic astigmatism*. The position of the axis should be noted, and convex cylinders of increasing strength successively inserted, preserving the same axial position so long as V is improved. If convex cylinders give no aid, concave cylinders should next be similarly tried.

The patient has *compound hypermetropic astigmatism*, *simple hypermetropic astigmatism*, or *mixed astigmatism*, according as the concave cylinder lens, if any, which gives the best vision has a numerical strength *less than*, *equal to*, or *greater than* the convex spherical glass with which it is combined.

If, however, a weak convex spherical glass impairs rather than improves visual acuity, it should be removed, and concave spherical glasses should be employed, commencing with the weakest, and gradually increasing in strength. If V is improved thereby the patient is evidently *myopic*, and the feeblest glass with which the greatest improvement in vision is obtained is the measure of the patient's spherical error.

Here again, if V be less than $\frac{6}{6}$, a cylinder should be placed in front of the spherical glass, commencing with a weak concave; the best position for the axis of the cylinder should be sought, and its strength gradually increased. If improved vision results, the patient has *compound myopic astigmatism*. If concave cylinders produce no benefit, try convex. The patient has *compound myopic astigmatism*, *simple myopic astigmatism*, or *mixed astigmatism*, according as this convex cylinder, if any, which gives the best vision, has a numerical strength *less than*, *equal to*, or *greater than*, the concave spherical glass with which it is combined.

In any case where spherical glasses alone have been rejected, *cylinders alone* should be tried. Indeed simple hypermetropic or simple myopic astigmatism is usually discovered in this way. But it sometimes happens, and especially in patients whose vision is not very acute, that the examiner is misled by the patient into believing that the eye requires the aid of a spherical glass, when in reality a simple cylinder is all that is needed, and this is ascertained by the patient admitting that he sees best when a cylinder of equal numerical strength, but opposite sign, has been combined with the spherical glass first adopted, and has neutralised its effect upon rays passing at right angles to the axis of the cylinder.

If the examination is made with the object of ordering glasses, it is well, after correcting each eye separately, to make a final test with both eyes open, in order to see that no diplopia or sloping of objects results from unequal focussing in the two eyes.

Now, though these details of the method of estimating the visual acuity have occupied some time to describe, with a little experience on the part of the observer, and a moderate degree of intelligence on the part of the patient, they can be very rapidly performed in practice.

The great advantage of commencing the investigation in this way is, that if no subjective visual sensations are complained of, and $V = \frac{6}{6}$ has been obtained in each eye with or without glasses, we may *generally* conclude that the sense of sight is normal, and further examination is needless, unless to exclude the possibility of congenital colour blindness or some ophthalmoscopic change being overlooked. The ophthalmoscopic examination (see under Objective Examination, p. 350) should never be omitted, since it occasionally happens that evidence of disease is found in the fundus, though the direct visual acuity is not affected. If, however, V cannot be raised above $\frac{6}{12}$ or $\frac{6}{18}$ even with glasses, a careful objective examination should be made, and unless a sufficient explanation of the defect is thus obtained, *e.g.*, high refractive error, irregular astigmatism, or corneal opacity, it will be well, especially if the defect is of recent origin, to determine the state of the colour sense and the extent of the fields of vision, before assuming that the eye is congenitally amblyopic.

In many cases, especially in adults, where $V = \frac{6}{24}$ to $\frac{6}{60}$ and cannot be improved by glasses, the physician may be enabled to form a sound diagnosis with rapidity by determining (1.) the power of the eye to perceive colours, especially in the central parts of the field (see p. 319); and (2.) the extent of the field of vision for hand movements (see p. 308), before proceeding to the ophthalmoscopic examination.

If a patient fail to see the largest type at 6 metres (*i.e.*, $V =$ less than $\frac{6}{60}$), he should be allowed to approach the card slowly until the largest types are seen. The distance must then be measured and expressed as the numerator, thus (say) $V = \frac{4}{60}$ or $V = \frac{1}{60}$, according as the individual is 4 metres or 1 metre from the type.

In no case should the expression $\frac{6}{0}$ be used to express $V =$ less than $\frac{6}{60}$, since $\frac{6}{0} =$ infinity, and while failing to express what is intended, gives no information as to the actual amount of the defect.

Counting fingers.—A rougher method of testing the acuity of vision when less than $\frac{6}{60}$ is that in which the observer holds up one or more of the fingers of his hand, invites the patient to count them, and in retiring from him varies the number of fingers so long as they are correctly counted or named. The result is expressed thus—"Counts fingers at (say) 3 metres." An observation such as this, though crude, is of value in estimating the further progress of a case.

Hand reflex.—If the patient cannot count fingers when brought within a few inches of his face, we proceed to see whether he can perceive an up and down or side to side movement of the hand held directly before his eye. If he can, we say that he has "hand reflex."

Perception of light.—So marked a depreciation of visual power always implies dense opacity in the media (*e.g.*, ripe cataract, hæmorrhage or exudation into the vitreous), detachment of retina, or serious failure of nerve power, or both, and in such cases it is always well at the same time to determine whether the perception of light is good, not only at the posterior pole of the eye, but over the general surface of the retina, and in those cases in which hand reflex cannot be elicited, this is the last step by which we ascertain whether the eye possesses any visual power or is altogether blind.

The simplest method is to make the patient face a bright window, exclude one eye completely, and place a hand a little in front of the one to be tested, so as to shade the light from it for a moment, then uncover and re-cover the eye, asking the patient to say if he notices any difference, when it is light and when dark.

Care should be taken that in making these movements the moving hand does not touch the face or give any other sensory indication of its change of position. The test may be more accurately applied in a darkened room by reflecting light with an ophthalmoscopic mirror, so as to enter the eye from successively different directions. The patient is requested to direct his gaze straight forward, to declare the moment he perceives any light on his eye, and to state whether it is coming from above or below, from his right or left side.

The strength of the light employed can be readily increased or diminished by varying the size of the flame employed or the distance from which it is reflected.

Having thus rapidly traversed the way by which we are led to determine the state of the *direct visual acuity for form* from perfection to blindness, we turn now to the other simple visual sensations.

The examination of the *sense of colour in direct vision* need only be undertaken in a special manner when there is any reason to suspect the presence of congenital colour blindness, and it then demands great care and discrimination in the method employed. Space does not permit of a proper presentation of this subject in a work of this kind, and the reader should consult the ophthalmic text-books of Berry, Juler, or Swanzy, or a special treatise as Joy Jeffries' on "Colour Blindness."

Some clinical means for testing colour perception, chiefly in indirect vision, will be referred to later.

II. The field of vision (*i.e.*, indirect vision) should now be examined.

There are several methods of different degrees of precision available for this purpose.

1. Estimation of the extent of the field of vision for *hand movements.*

This is a very rough but exceedingly useful test where the central acuity has been found markedly defective.

Place the patient with his back to the window or source of light; make him cover one eye lightly; stand directly opposite him at a distance of two feet, and close the opposite named eye in your own person. Then let each individual gaze at the other's open eye. The field of vision of each should now be identical, though seen from opposite aspects. The observer with a healthy eye has thus a ready means of comparing the extent of the patient's power of perception with his own, while his outstretched hand moves radially outwards from or inwards towards the common visual axis, in different directions, but always in a vertical plane midway between his own and the patient's eye.

A decided contraction of the field, as is met with in *e.g.* optic atrophy or retinitis pigmentosa, or the position of an extensive scotoma, as from detachment of the retina or choroido-retinal atrophy, may be rapidly determined by this method.

2. By the use of a large black board the field of vision may be projected on to a plane surface.

Such an arrangement is called a *campimeter*.

The patient should stand with his back to the window, cover one eye, and fix the gaze of the other on a small white spot affixed to the centre of the board, which should be directly in front and eighteen inches from his face. Various test objects can be affixed in turn to the end of a long black rod, and moved radially outwards from the fixation point, or inwards from the periphery, and the remotest point in each direction at which the object is perceived is marked on the surface of the board in chalk. A piece of white chalk makes a convenient test object and marker combined.

A line connecting all the chalk marks, of any one colour, gives an outline enclosing the field of vision as thus obtained for that colour.

But this method is not well suited for the peripheral parts of the field, since in order that these may be tested the object must be removed further from the eye, and the test becomes unequal. For the more central parts it is practically accurate in principle, and may be very conveniently employed in a manner which will be explained later on (see p. 319).

3. *Perimetry* affords the most exact method of estimating the field of vision.

Several perimeters have been devised, differing in mechanical detail, but most consist of a graduated bar (semicircular or forming the quadrant of a circle) capable of supporting a suitable test object, and of describing the circumference of a hemisphere, whose pole—the pivot of the bar—is the point of fixation.

The eye to be examined should occupy the centre of the sphere, and thus the chief defect of the campimeter is overcome, for whatever the position of the test object on the bar, its radial distance from the eye, the size of its image on the retina, and its luminosity, are constant.

The straight line which connects the fovea centralis of the retina with the point of fixation is called the *visual axis.*

Any other straight line passing into the eye from any point on the quadrant to the retina must cross the visual axis at a certain angle, which increases directly as the distance of this point from the point of fixation.

This relation is expressed on the quadrant, which is divided into degrees, starting from 0° at the pivot (point of fixation) to 90° at the free end.

If the quadrant be set in rotation every point of it will describe a circle, and all the circles are concentric. The combined circles forming the imaginary hemisphere on which the field of vision is to be mapped out may be represented on a plane surface by a few of these circles described at equal angular intervals ; lines radiating at equal intervals from their common centre will represent meridians of curvature of the hemisphere. (See Fig. 97).

Such a representation, reduced in size, affords a chart which is usually affixed to the back of the perimeter.

The test object is a disc or 1·5 centimetre square of white or coloured unglazed paper. If inserted into the end of a small black rod (6 or 8 inches long) it can be held by the physician in any desired position, or it may be supported in a clip attached to, but moveable, along the concave aspect of the quadrant.

The observation should be made in good diffuse daylight.

Let the patient sit with his back to the window. Lightly bandage one eye or cover it with a small shade, taking care that no part of the obscurer is visible in, and therefore does not curtail, the field of the other eye. Place the perimeter on a table in front of the patient, and carefully adjust its height so that the fixing eye can be maintained without discomfort in the same horizontal plane as the point of fixation. To ensure steadiness a head-rest for the cheek or chin is usually supplied.

The physician, facing the patient, directs him to fix his gaze on a small white spot at zero on the quadrant, and as the examination proceeds takes care to recall the patient's eye if it wanders from the fixing point.

Having set the quadrant in some definite meridian—say the horizontal—the test object is brought slowly along the arc from the periphery towards the centre. The patient is to declare the moment at which he first perceives its advance, and to state its colour if coloured. In the latter case especially he may have some hesitation in deciding, and if so, the direction should be reversed, and the limit fixed approximately by enquiring for the point at which he appears to lose the colour after clearly perceiving it in the more central part of the field.

The physician having recorded this by a prick or coloured pencil mark on the corresponding spot of the chart, turns the quadrant through a few degrees, repeats the observation in another meridian, and again records the result.

The greater the number of meridians examined, the more complete the information derived; but it must be remembered that if the examination be prolonged, fallacies may arise from exhaustion of the retina. A curved line connecting similar marks on the meridians examined gives the outline of the field for the corresponding test object employed.

In moderate daylight the fields for white and black are larger than those for any of the colour tones, but Landolt has shown that in a normal eye, if the illumination be sufficiently bright, all the fields are co-extensive.

The accompanying chart shows the oval shape of the fields, that their greatest expanse is outwards and downwards, least upwards and inwards. The curtailment of the latter is chiefly due to the projection of the nose and brow.

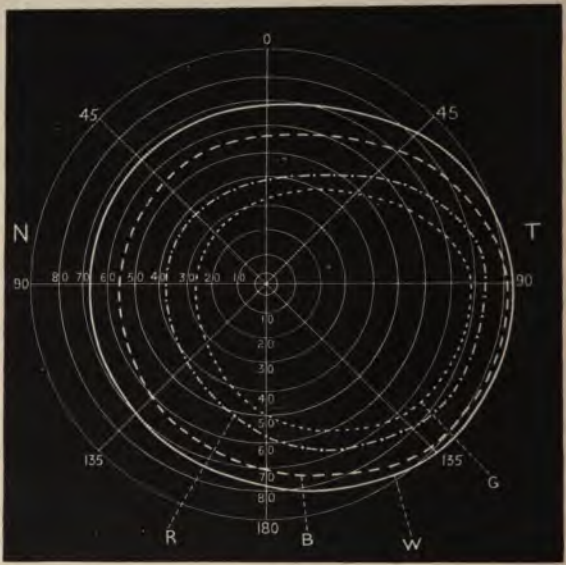

Fig. 97.—Perimetric chart showing field of vision of right eye—for white (*W.*), blue (*B.*), red (*R.*), and green (*G.*), as projected on the inner surface of a hemisphere whose pole is the point of fixation. *N.* denotes the nasal or inner, *T.* the temporal or outer half of the field.—(Landolt.)

In practice it is convenient to take first the field for white, which alone may give sufficient evidence to warrant a diagnosis, and avoids the fallacy of any congenital defect for colours.

In the selection of colour tests, care should be taken that the colours employed are as pure as can be obtained.

Red and green form one pair of complementary colours, blue and yellow another pair. Under ordinary illumination the latter have the larger fields.

It is evident that if either the red or green test object contains much admixture of yellow or blue, a considerable error in the apparent size of the fields may be introduced. The presence of a decided impurity in a colour test may be readily detected by the examiner, if his own colour sense be good, by noting whether the test object, which appears red or green when viewed directly, becomes bluish or yellowish when viewed by himself indirectly in the inner half of the field of either eye, and moved into an increasingly peripheral position.

The fields of vision may be found defective—

(*a.*) In extent or continuity.
(*b.*) In acuity.

(*a.*) Concentric contraction of the fields is met with notably in retinitis pigmentosa, and usually in atrophy of the optic nerve; but not infrequently in the latter the shrinking of the field is irregular, depending doubtless upon the order in which the process affects the nerve bundles.

In chronic glaucoma there is also gradual diminution of the field, but the inner limits are usually first curtailed, and the outer shrinks more slowly.

In embolism of the central artery of the retina, if any vision is retained at all, it is in the outer part of the field, and there seldom exceeds the perception of hand movements. Embolism of a branch of the same artery will give rise to a failure in that segment of the field which corresponds to the impoverished retinal area. In detachment of the retina, in atrophy of the retina from injury or disease of the adjacent choroid, and usually in congenital coloboma of the choroid, a defect in the field will be found corresponding to the lesion as determined by the ophthalmoscope.

In some cases, though the fields are of normal *extent*, a localised area of blindness, a scotoma, may be found interfering with their *continuity*.

The optic disc is a physiological scotoma about 15° to the outer side of and below the point of fixation.

Scotomata may be small or large, single or multiple. They are described as *positive* if the patient is conscious of their interference in his field, *negative* if, like the optic disc, they do not obtrude themselves upon his consciousness.

In the latter case they may not be discovered by the physician unless he takes the precaution of ascertaining not only the outlines of the fields, but also whether the test object is properly perceived along each meridian, from the limit inwards to the fixing point.

In mapping out a scotoma it is often a good plan to attach the test object to the quadrant and slowly rotate it through as many degrees to the right or left as may be necessary to determine where it can and where it cannot be perceived by the patient. By combining circular movements of this kind with the to-and-fro radial movements along the quadrant previously described, the dimensions of the scotoma may be pretty accurately demarcated.

As already indicated (see p. 310) every ray which enters the eye, except that coming from the point of fixation, crosses the visual axis before it reaches the retina.

[*Note.*—The point of intersection of all the entrant rays is the nodal point of the eye. It is situated close behind the crystalline lens in the anterior part of the vitreous.]

Consequently rays coming from the right half of each field are focussed on the left half of each retina, and similarly with the right half of each retina we perceive objects situated in the left half of each field, so far at least as the fields are coincident; objects situated above the visual axis are focussed on the lower part of the retina, while those below the line of fixation imprint their images on the upper part of the retina.

Clinical and pathological observation have combined to prove that the right half of each retina is innervated by the nerve fibres which compose the chief part of the right

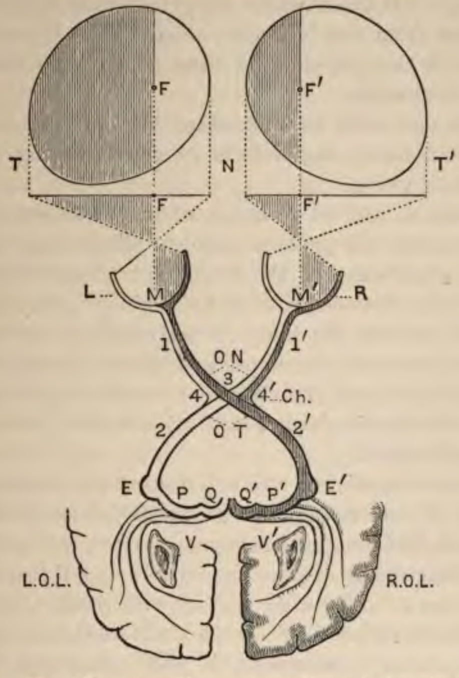

FIG. 98.—Diagrammatic representation of the visual conducting paths and fields of vision, to show the crossing of the rays in the media, and the relation of the half-fields to the eyes and to the cerebrum.

L. Left eye. *R.* Right eye.
M. & *M′.* Macula lutea in each.
F. & *F′.* Point of fixation.
(In binocular vision *F.* & *F′.* coincide, and the fields overlap.)
N. Between the nasal or inner half-fields.
T. & *T′.* The temporal or outer half-fields.
O. N. Optic nerves. *Ch.* Chiasma.
O. T. Optic tracts.
E. E′. External geniculate body.
P. P′. Pulvinar (posterior end of optic thalamus).
Q. Q′. Corpora quadrigemina.
V. V′. Posterior ends of lateral ventricles.
L. O. L. Left occipital. *R. O. L.* Right occipital lobe.
For 1, 1′, 2, 2′, 3, 4, 4′, see text.

optic tract, while those which supply the left half of each retina come from the left optic tract. The inner half of each retina is thus supplied by fibres which have decussated at the optic chiasma.

We are now able to understand the occurrence of symmetrical scotomata, *i.e.*, defects in corresponding parts of both fields of vision.

For while a destructive lesion of one optic nerve, as at 1 or 1′ in Fig. 98, will produce complete blindness of the corresponding eye, a lesion of the left optic tract at 2 will cut off communication between the *left half of each retina* and the brain, and produce blindness in the *right half of each field* (right homonymous hemianopsia), similarly a lesion at 2′ will render the *right half of each retina* functionally useless and produce blindness in the *left half of each field* (left homonymous hemianopsia).

A lesion in the chiasma at 3 will destroy the function of the *inner half of each retina*, and produce blindness in the *outer half of each field* (temporal hemianopsia); while it will require a very symmetrically disposed lesion at 4 and 4′ to cut off the sensory fibres from the *outer half of each retina* and produce blindness in the *inner half of each field* (nasal hemianopsia).

Homonymous hemianopsia is the commonest variety; temporal hemianopsia is fairly frequent; but only one authentic case of nasal hemianopsia appears to have been described, and considering the peculiar lesion required, its uniqueness is not surprising.

Superior and inferior hemianopsia, *i.e.*, defective vision in the upper and lower halves of the fields respectively, have been described, but are much more likely to be due to retinal disease, *e.g.*, detachment of the retina in the same part of each eye, than to symmetrical lesions in the course of the nerve trunks.

In no case of supposed hemianopsia should a diagnosis be made without a very careful objective examination. If the entire half field of each eye is functionless the hemianopsia is said to be *complete.* If part of it retains visual power the case is *incomplete.* In the latter case the scotoma may be sector-like, or insular, or quite irregular.

It is by no means easy to determine the exact seat of a lesion producing lateral homonymous hemianopsia, since it may be on the optic tract only a short distance behind the chiasma, or nearer the basal ganglia, or above these in the occipital cerebral substance as high as the occipital cortex. There is, however, one objective test associated with the name of Wernicke, which, though of doubtful value, we may conveniently refer to here.

It depends on the supposition that there is a special set of nerve fibres concerned purely in the conduction of the stimulus of light from the retina to the third nerve nucleus in the floor of the aqueduct of Sylvius, that these fibres do not convey *visual sensations,* but leave the optic tract at the level of the basal ganglia, and do not pass up to the occipital cortex.

Wernicke suggested that if the lesion causing hemianopsia was situated above the point of emergence of these pupillary light-reflex fibres, the pupillary reaction to light ought to be carried on perfectly, though the light stimulus was applied only to the blind half of the retina. But if the lesion occurred below the point of emergence of the said fibres, the careful admission of light into the eye in such a way as to fall only on the blind half of either retina ought to be followed by no contraction of the pupil.

Seguin recommends that the light should be thrown into the eye from an ordinary ophthalmoscopic mirror at an angle of 40° to 60° from the visual axis. It is evident that the test can be of no service unless the tract lesion is *absolutely destructive* in its effects on the fibres which conduct visual sensations. This leads us to the second aspect in which the fields of vision may show deficiency, namely :—

(*b.*) *Acuity.*—There may be various grades of impairment of visual acuity. Speaking generally, it would appear that whenever visual failure is essentially due to disease of the optic nerves or tracts, the sense in which the failure is earliest exhibited is that of colour, and notably in the perception of red and green. In slowly advancing optic atrophy blue and yellow may still be perceived throughout small fields, when red and green are no longer perceptible under ordinary illumination.

It is probable, too, that in all cases the sense of form for objects seen indirectly or directly fails in some proportion to the colour sense, but the peripheral form sense has never received much attention. It may be tested readily by employing strips of white cardboard having one, two, or three black squares (2 centimetres square) respectively placed on them at equal intervals, the patient being requested to tell how many squares are visible on any one exposed strip, and the moment at which they come into view when brought along the quadrant of the perimeter.

We have as yet no satisfactory method of testing the light sense (in the exact meaning of that term) in indirect vision, but it is scarcely necessary to point out that after all vision for white and colours has disappeared in any scotomatous area, and when even hand movements are no longer perceived, some perception of bright light may still be retained.

In hemianopsia it is usual to find that all the senses are greatly subdued. If no perception of light remains in the affected half fields the hemianopsia is said to be *absolute*; and the same adjective may be applied to any other form of scotoma which is entirely destitute of sensation.

If, however, some perception remains, the defect is said to be *partial*.

Some disturbances of the field of vision which affect the more central parts of the field, and are most readily detected by a colour test, still remain to be mentioned.

In practice these may be diagnosed so much more expeditiously with a small campimeter rather than a perimeter, that we shall conclude this section with a brief account of this simple apparatus and its use.

The Examination of the Colour Sense in the Peri-Central parts of the Retina.

For this purpose a piece of stiff black pasteboard about 18 inches square may be conveniently employed, having a small white spot marked at the centre of its surface.

A narrow strip of similar material, 6 or 8 inches long, should also be prepared, with a small disc of one of the fundamental colours (red, green, blue, and yellow) affixed near the free end of each surface.

The patient stands with his back to the window. The physician, facing him at a distance of two feet, holds the board in one hand against his own breast, requests the patient to cover one eye and fix the gaze of the other on the central white spot.

Taking the colour-bearer in his free hand, the physician quickly applies one end to the centre of the board, so that the white spot is now replaced by a coloured disc of (say) red, and invites the patient to name it, or at least to say whether it appears grey or has any colour. Whether the reply be satisfactory or not the disc should next be moved radially outwards in different directions, care being taken that the patient's eye does not follow it nor wander from the fixing-point. Now, if in any eccentric position the colour is more clearly perceived than at the centre, the colour sense at the macular region is certainly at fault, since normally it is most acute at this part, and provided ophthalmoscopic examination reveals later no opacity in the media nor visible change in the fundus to account for it, we may conclude that the nerve fibres supplying the fovea centralis are morbidly affected at some part of their course.

With a little care the extent and shape of the colour-scotoma may be mapped out.

If central, involving the point of fixation and its immediate neighbourhood irregularly, but extending chiefly to the inner side (nasal side of field), the case is probably one of inflammation affecting the optic nerve in the orbit—retrobulbar neuritis.

If extending chiefly to the outer side of the fixing-point, and corresponding to (*i.e.*, subtending) that part of the retina between the optic disc and macula, known as the "papillo-macular area," the case is almost certainly one of toxic amblyopia, due to the abuse of tobacco, and perhaps alcohol.

Retrobulbar neuritis usually affects only one eye, while toxic amblyopia commonly, if not always, affects both, and the scotoma is symmetrical.

The test should be made with red and green, for blue and yellow are generally well perceived, except in severe cases.

It should be borne in mind that *congenital colour blindness* affects from two per cent. to three per cent. of the male population of this country, and a less proportion of females.

It is commonest for red and green, but may be distinguished from the foregoing by the fact that *per se* it in no way affects the direct visual acuity for form as tested, *e.g.*, by Snellen's types.

If, however, in a congenitally colour blind person from any cause the form sense has been rendered defective, a proper diagnosis might still be made by remembering that congenital colour blindness affects the whole field, while in the above affections, if uncomplicated, good colour perception is retained beyond the limits of the scotoma.

With this simple apparatus the various forms of hemianopsia, if approximately complete in extent, may be also readily diagnosed, and practically it is only when we wish to determine precisely the state of the peripheral parts of the fields for different colours, or require a permanent record, that we need have recourse to perimetry.

Phosphenes.—This term is applied to the visual sensations induced by mechanical stimulation of the retina,—*e.g.*, the pressure of the tip of the little finger on the outer side of the globe occasions the sensation of a bright crescent with a dark centre in the inner part of the field.

In an intelligent individual it is possible to make use of this as a test of the retinal capacity for stimulation when *from any* cause it is not possible to see the fundus clearly.

THE SENSE OF HEARING.

The first object of the physician is to test the amount of hearing power actually present. This is done by two methods, both of which should be employed in every case so far as practicable.

(1.) *The Watch.*—For this purpose a watch is used which has been tested in a number of healthy individuals. Let us assume that the one employed is normally heard at 30 inches. It is put at this distance from the patient's ear and gradually approached until the tick is perceived. Assuming, for the sake of demonstration, that the distance is then 12 inches the hearing power may be conveniently expressed as $\frac{12}{30}$.

(2.) *Conversation.*—It is next necessary to test the power of hearing conversation. This is best done by directing the patient to sit with the ear it is desired to examine towards the experimenter, while the other is occluded by the finger. Words are then whispered or spoken as the physician gradually approaches until they are heard and repeated. It is thus noted what is the distance at which whispered or ordinary conversation is heard. Sometimes only a loud voice near the ear is perceived, and occasionally not even that.

The observer next proceeds to employ the *tuning-fork* test. The vibrating fork is applied to the middle line of the forehead, and the patient questioned as to which ear perceives the vibrations more distinctly. The whole question as to the value of the tuning-fork test must be at present considered *sub judice*. As a general rule, however, *the tuning-fork applied in this way is heard more in the deaf ear if the lesion causing the deafness be in the meatus or middle ear, and less if the labyrinth or auditory nerve be involved.* A considerable number of exceptions to this rule are, however, met with. Another method is to apply the turning-fork over the mastoid of the affected ear, and immediately it ceases to be perceived bring it before the meatus. *In cases of nerve deafness as a rule the fork is better heard by air than by bone conduction.*

SENSE OF TASTE.

The sense of taste is somewhat complex. The posterior third of the tongue with the fauces and pharynx are brought into connection with the centres by means of the glosso-pharyngeal nerves; the anterior two-thirds are supplied by the lingual nerve; and the sensory branches concerned in taste appear to pass into the *chorda tympani*, whence by its connections they proceed to the trigeminal or glosso-pharyngeal nerve—it is not yet certain which—and reach the brain.

Subjective perversions of the sense of taste are rarely met with except in mental disease.

To test the sense of taste substances devoid of aroma should be employed—such as sweet, bitter, sour, and salt bodies. They should be applied to each half of the tongue, both in its anterior and posterior parts.

Increased sensibility is very rare except in hysterical patients.

Diminished sensibility may be due to central or peripheral lesions, and the extent of the anæsthesia points to the locality of the disease. For instance, if there be loss of taste in the anterior part of the tongue, while the patient has no loss of ordinary sensibility in the tongue, the *chorda tympani* is in fault. If there be loss of the sense of touch as well as of the sense of taste, it is the *lingual* which is affected. The attendant symptoms enable the observer to estimate the level at which the lesion is situated.

2.—MOTOR FUNCTIONS.

In an investigation of the motor functions it is necessary to examine the tone and size, the voluntary and involuntary movements, as well as the irritability, of the muscles tested by mechanical and electrical stimuli.

(*a.*) The *tone* of the muscles may be estimated by the rigidity or flaccidity which is felt by the hand on grasping them. Experience teaches the observer to recognise the normal consistence of a muscle, and to detect any deviations from it.

Abnormal *flaccidity* of a muscle or group of muscles follows the removal of motor influences in any part of the nervous tracts.

Abnormal *rigidity* may be the result of two different classes of affections. It may be due to irritation of the motor tract, as in cases of sclerosis of the pyramidal tracts, or it may be caused by disease of joint or muscle, involving the retention for a long time of a definite posture. This condition is commonly known as *contracture.*

(*b.*) The *size* of the muscles may be estimated by the outline which they present, or may be more accurately ascertained by the measuring tape. Alterations in size may be produced by nervous diseases, or by other affections involving their lessened employment.

A real *increase* in the size of any muscle or group of muscles is only caused by continuous use. An apparent increase is seen in the condition known as pseudo-hypertrophic paralysis.

A *diminution* in the bulk of any muscle or group of muscles may be the result of disuse simply. The muscles of a limb, for example, which has been rendered immoveable by ankylosis of a joint, invariably undergo a certain amount of atrophy.

Rapid wasting of muscles is in all cases produced by affections involving the loss of the influence of the anterior grey horns of the cord. It is found in disease of the motor nerves, or of the anterior nerve roots, or of the anterior cornua of the cord.

(*c.*) The *movements of the muscles* may be modified in different degrees, from slight impairment to entire abolition. They may be tested by causing the patient to exert pressure against an obstacle such as the hand of the observer. Various instruments—such as the dynamometer—have been invented in order to test the strength of the muscles, but none of these are so satisfactory as the comparison of the power of different muscles by the hand itself.

When motor functions are lost, the term *paralysis* is employed, and it is at present fashionable to denote slighter degrees of impairment by the phrase *paresis*. As there is a perfect gradation between the different degrees of diminished function, the use of different terms for the same condition is to be deprecated.

Certain terms are used to indicate the extent of the paralysis. In cases where only one group of muscles is paralysed, the term *monoplegia* is employed; where one lateral half of the body is paralysed, the case is said to present *hemiplegia;* and where both sides are paralysed below a certain level, the symptom is called *paraplegia.*

The exact site of a lesion causing paralysis is to be inferred from the position and extent of the effects, as will be further insisted on in the section dealing with reflex action. In this place it will be well to mention that in an affection of an *ordinary spinal nerve* both motor and sensory functions are interfered with in one region, and there is rapid wasting; in a *total transverse spinal lesion* both of these functions are also disturbed, on both sides of the body below the level of the lesion, and there is rapid wasting of a small area corresponding to the anterior cornual zone affected; in affections of the *anterior grey matter* of the cord, the paralysis is attended by rapid wasting; in disorders of the *motor tract,* the paralysis is associated with spasmodic symptoms, and if the motor tract be diseased in the medulla oblongata, there is frequently some interference with such organic functions, as respiration, or circulation, or deglutition; in diseases situated in the *anterior part of the pons cerebri* there is paralysis of one side of the body and of the opposite side of the face, commonly termed "crossed paralysis;" if the *crus cerebri* is destroyed, there is paralysis of one side of the body, and of some of the ocular muscles of the other side; when there is a lesion in the *anterior part of the internal capsule* there is purely motor paralysis, often with contracture as a sequel, and if the disease is in the *posterior part* there is paralysis of motility and sensibility.

(*d.*) *Abnormal movements* may be observed in muscles. These are usually termed *spasms*. Spasms are of two kinds:—*clonic*, in which there is alternate contraction and relaxation; and *tonic*, in which there is persistent contraction of a muscle or group of muscles.

Clonic spasms vary in extent and degree, from faint movements in muscular fasciculi, causing no change in the position of any limb, to general convulsions, involving the entire body. Minor degrees are termed *tremors*, and the slightest form is the *fibrillary contraction* of individual muscular bundles, causing a faint wave to travel along the surface of a muscle. On a larger scale they may be seen in the rhythmic tremors, which are present both during repose and action in paralysis agitans, in the irregular jerks seen in chorea during rest and activity, and in disseminated sclerosis during the action of the muscles. In the highest development they form the local or general convulsions characteristic of explosive nervous diseases. A special form of clonic movement is seen in athetosis, which usually occurs as a sequel to hemiplegia. In this symptom the fingers and toes maintain a series of continuous slow movements.

Tonic spasms likewise vary in extent and degree. In their slightest form they occur in cramp, affecting individual muscles, in a more pronounced form as contracture, and in their highest possibility as tetanus.

(*e.*) The *mechanical irritability* of a muscle is easily tested by the application of such an excitant as a moderately strong blow, which under ordinary circumstances produces a contraction of the part of the muscle struck. Changes in the degree of mechanical irritability are of small aid to diagnosis. In certain wasting diseases, especially phthisis, the irritability is greatly increased.

(*f.*) The examination of the *electrical irritability* is of great value in the diagnosis of nervous diseases.

Two forms of current electricity are employed in modern medicine—the *primary* or *galvanic*, and the *induced* or *faradic* current. The former is employed as a continuous or slowly interrupted current; the latter from its nature is always used as a very rapidly interrupted current.

The *continuous current* produces no muscular contraction when flowing, but causes contraction when closed or opened. The cathode or negative pole chiefly produces contraction when the current is closed; the anode or positive pole when the current is opened; and the cathode produces stronger effects than the anode.

The *law of normal galvanic irritability* may be stated here as a formula. Let K = cathode, A = anode, C = closure of current, O = opening of current, $\mathrm{C}-$ = weak contraction, C = moderate contraction, $\mathrm{C}+$ = strong contraction, and C^t = tetanic contraction; then—

Weak currents give $\mathrm{K}C\mathrm{C}-$;
Medium currents give $\mathrm{K}C\mathrm{C}+$, $\mathrm{A}C\mathrm{C}-$, $\mathrm{A}O\mathrm{C}-$
Strong currents give $\mathrm{K}C\mathrm{C}^t$, $\mathrm{A}C\mathrm{C}$, $\mathrm{A}O\mathrm{C}$, $\mathrm{K}O\mathrm{C}-$

In other words, weak currents produce slight contraction at the cathode on closing the circuit, but no contraction at the anode.

Medium currents produce strong contraction at the cathode on closing, and slight contraction at the anode both on closing and opening the circuit.

Strong currents produce tetanic contraction at the cathode on closing, moderate contraction at the anode both on closing and opening, and slight contraction at the cathode on opening the circuit.

The *induced current* produces contraction of muscle when applied directly to it, or indirectly through the nerve of supply.

The *law of normal faradic irritability* is that the degree of contraction varies directly with the strength of the current employed.

Changes in the galvanic and faradic irritability are characteristic of different classes of nervous affections.

A. *Normal irritability.*—In hysterical and most cases of spastic paralyses there is no alteration in the galvanic or faradic irritability.

B. *Quantitative alterations of irritability.*—The change may be of the nature of an increase or decrease.

An *increase* of galvanic irritability is found in some cases of early hemiplegia, of spastic paralysis, and of locomotor ataxy. This causes some modifications from the conditions stated as the law of irritability, inasmuch as currents produce much more powerful effects than under normal circumstances.

A *decrease* occurs in the later stages of all paralysis leading to wasting of muscles, and it is found in all muscles which have undergone atrophy from disuse.

C. *Qualitative and quantitative alterations of irritability.*—These changes are characteristic of cases in which there is great disturbance of the nutrition of muscles. They are found in the various forms of peripheral paralysis from inflammatory changes in, or the effects of toxic agencies on, nerve trunks. They are also present in diseases of the anterior cornua of the cord and the corresponding structures of the medulla. They are not met with in cases of cerebral paralysis, of spastic paralysis, of hysterical paralysis, or of paralysis from disuse of muscles. The great use of these alterations is the distinction given between central and peripheral lesions.

The alterations are commonly known by the designation of *the reaction of degeneration.* As they do not follow a similar course in nerve and muscle, these must be taken up separately.

The reaction of affected *nerves* is marked by a progressive lessening of both galvanic and faradic irritability, without any alteration in their mutual relations to each other. This goes on in cases which are incurable to a permanent loss, but in curable cases the irritability to both currents returns simultaneously and gradually.

The reaction of the affected *muscles* to the different currents is marked by a change in their relationship. The galvanic irritability lessens for a few days after the commencement of the attack, and after ten or twelve days becomes greatly increased, so that the muscles respond to weaker currents than in health. This continues until about the fourth week, from which a gradual lessening goes on in curable cases to the normal, in incurable cases to a total loss. There is also a change in the relations of contraction at the cathode and anode, so that the anodal closing contraction is as great as, or greater than, the cathodal closing contraction, and the cathodal opening contraction is as great as, or greater than, the anodal opening contraction. The faradic irritability is gradually lost, just as in the case of affected nerves, and gradually regained in curable cases.

In employing any form of battery, the skin and electrodes should be well moistened before testing irritability, and the weakest current that will produce the results desired should be used. That pole of the battery whose action is to be determined should be placed over the region to be tested, and the other on some distant spot. For further information in regard to the use of electricity in diagnosis as well as of the motor points—that is, the spots where nerves enter muscles—special treatises on the subject must be consulted.

3.—Reflex Functions.

Reflex irritability affords useful aid in diagnosis. The explanation of the phenomena to be mentioned in this place cannot be said to be quite satisfactory, but it may be said that for the performance of any reflex act there must be unimpaired activity of the different structures entering into the reflex loop. Under the head of reflexes must be included the *superficial* and the *deep*.

The *superficial reflexes* are numerous and are produced by gentle stimulation of the skin, which induces contraction of certain groups of muscles. The afferent impulses proceed to the centre by the nerves of ordinary sensation ; the efferrent impulses pass outwards by the motor paths. They are named from below upwards the *plantar*, *gluteal*, *cremasteric*, *abdominal*, *epigastric*, and *scapular*. They require no description, as the regions of the skin to be stimulated are shown on the accompanying table from Gowers, along with the muscles and nervous centres concerned in each. By their means the condition of almost the entire extent of the spinal cord may be ascertained. The superficial reflexes are *increased* in cases where the spinal cord is in a state of exalted irritability, such as tetanus and strychnine poisoning, or where the cerebral inhibitory action is reduced, as in some cases of cerebral or spinal paralysis. They are *diminished*, on the other hand, when there is any interference with the reflex loop, such as disease of the nerves or of the cord itself, and where the cerebral inhibitory influences are increased, as in certain cases of irritative cerebral disease.

In additional to these superficial reflexes, there are the *cranial reflexes*, as they are termed by Ross. The principal of these are the sneezing produced by irritation of the nose; the closure of the iris produced by light; the winking produced by irritation of the conjunctiva, or the approach of some object; the muscular contractions produced by a loud noise; the contraction of the muscles of the palate and gullet produced by irritation of the fauces; and the coughing produced by irritation of the larynx.

One of the most interesting modifications of these cranial reflexes is the failure of the iris to react to light, while it undergoes the usual changes on looking at near and far objects. This shows that while the reflex action has been lost, the associated movement remains. This interesting symptom is often found in locomotor ataxy, and is named the *Argyll Robertson phenomenon*, in honour of its first observer.

The *deep reflexes* are of even greater importance than the superficial. They are excited by stimuli applied to deeper structures than the skin, such as muscles and tendons, but

TABLE showing the APPROXIMATE RELATION to the SPINAL NERVES of the various SENSORY and REFLEX FUNCTIONS of the SPINAL CORD. (*After* GOWERS.)

	MOTOR.	SENSORY.	REFLEX.
1. C.			
2.	Sterno-mastoid,	Neck and scalp.	
3.	Trapezius,		
4.	Diaphragm,	Neck and shoulder.	
5.	Serratus,	Shoulder,	
6.	Shoulder,		
7.	Arm,	Arm,	Scapular.
8.	Hand,	Hand,	
1. D.			
2.			
3.			
4.			
5.	Intercostal muscles,	Front of thorax,	Epigastric.
6.			
7.		Ensiform area,	
8.			
9.			
10.	Abdominal muscles,	Abdomen,	Abdominal.
11.		Buttock, upper part,	
12.			
1. L.			
2.	Flexors, hip,	Groin and scrotum (front),	Cremasteric.
3.	Extensors, knee,	Thigh { Outer side, Front ,, Inner side, }	Knee-jerk.
4.	Adductors, Hip,		
5.	Abductors, Hip,	Leg, inner side,	
1. S.	Extensors (?) Hip,	Buttock, lower part,	Gluteal.
2.	Flexors, knee (?)	Back of thigh,	
3.	Muscles of leg moving foot,	Leg and foot, except inner part,	Foot-clonus. Plantar.
4.	Perineal and anal muscles,	Perineum and anus.	
5. Co.	. . . . .	Skin from coccyx to anus.	

the explanations given of them at present leave much to be desired. Of the deep reflexes there are only two which merit notice—the *knee-jerk* and the *ankle-clonus.*

The *knee-jerk* can be produced under ordinary circumstances with comparative ease. If the knee is flexed and the leg allowed to hang freely, a tap upon the patellar tendon will be followed by a distinct jerk forward of the leg and foot from sudden contraction of the quadriceps muscle. The same result may in many cases be obtained by tapping the attachments of the quadriceps to the patella, or even by striking the muscle itself.

In order to study this phenomenon the patient should be placed in a sitting posture, and the observer should support the thigh of the patient by passing his arm beneath it so as to rest his hand on the other knee of the patient. The stimulus should be applied by striking the patellar tendon with the inner edge of the extended hand, with the margin of the large end of a stethoscope, or with a plessor. Care must be taken not to mistake mere swinging of the limb for the true knee-jerk.

The *ankle-clonus* may be produced in certain cases. In such cases, if the limb is extended nearly to the full, and the foot is passively flexed by the observer pressing the sole upwards by the application of the hand, a series of jerks or clonic spasms is produced by alternate contraction and relaxation of the muscles of the leg, which continues as long as the pressure is maintained. In order to produce the phenomenon it is well that the knee should not be entirely extended, but kept in a semi-flexed position.

The knee-jerk is present in almost all cases of health; the ankle-clonus is never found in healthy conditions. The extent of the knee-jerk varies greatly in health, and the ordinary amplitude of movement can only be ascertained by careful examination of different individuals, so as to learn what is to be regarded as an increase or decrease of the reflex. The significance of the ankle-clonus is more precise, as its presence is always pathological and speaks for an increase of the reflexes.

An *increase* of the deep reflexes is the result of exalted irritability of the grey matter of the cord, or of diminution of the inhibitory influence of the cerebral centres, as in hemiplegia, and especially in sclerosis of the lateral columns.

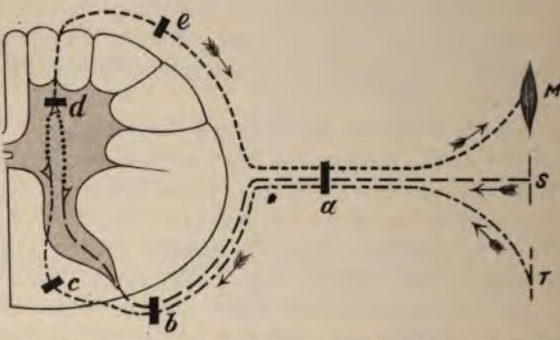

FIG. 99.—Diagram showing seats of interference with deep reflexes. M = muscle; S = skin; T = tendon.

A *decrease* of the knee-jerk is due to some interference with the reflex loop. This, as is shown by the diagram, Fig. 99, may be produced by disease of the nerve trunk (*a*), when motor and sensory functions are interfered with; by disease of the posterior root (*b*), when sensory functions only are altered; by disease of the posterior root zone (*c*), when no sensory or motor changes occur; or by disease of the anterior cornu (*d*) or anterior root (*e*), when motor functions are abolished and there is rapid wasting. It is most frequently diminished by disease of the posterior nerve roots, or of the posterior root zones, or of the anterior cornua.

4.—CO-ORDINATING FUNCTIONS.

The *co-ordination of muscular movements* is closely associated with the condition of the muscular sense, and is at the same time dependent on the integrity of the reflexes. The sensory and motor paths provide for impulses to and from the centres in the cord, which are probably under the influence of a higher centre, or of higher centres, in the ganglia at the base of the brain. The integrity of these structures provides for the perfect co-ordination of all muscular acts, and disease results in inco-ordination.

The power of co-ordination is to be *tested* by making the patient perform different movements, and observing the manner of their performance. In regard to the lower extremities, the patient may be directed to stand with his feet close together and his eyes shut, to walk along a line with his eyes shut as well as open, and to walk backwards.

Impairment of co-ordination is specially characteristic of locomotor ataxy, in which it is due to disease of the posterior root zone of the cord.

Closely allied in some respects to disorders of co-ordination are disturbances of the *balancing power* caused by interference with the centres concerned in the maintenance of the erect posture. In such cases the gait is staggering, and the patient cannot stand with his feet close together. This symptom differs from that above mentioned in the fact that while the patient is in bed he can perform all kinds of movements without apparent loss of the power of co-ordinating muscular movements, and further in the fact that there is no alteration at any time in the movements of the arms. Disturbances of the balancing power are found specially in disease of the middle lobe of the cerebellum.

5.—VISCERAL FUNCTIONS.

The most important visceral functions which may be disordered in affections of the nervous system are those connected with the circulation and the respiration, whose centres are situated in the medulla oblongata, as well as those concerned in defæcation, micturition, and generation, which have their centres in the lumbar enlargement. All these functions are frequently classed under the term *organic reflexes.*

The *circulatory functions* undergo alteration for the most part in changes of the rate and rhythm of the heart's action. As a result of cerebral hæmorrhage, for instance, a frequent pulse is often associated with a high temperature, while in consequence of a tumour or other intracranial affection the pulse may be very considerably retarded. Various forms of irregularity of the pulse are found in cerebral disorders, such as meningitis. All these symptoms are most marked when the disease is situated at the base of the brain near the medulla oblongata.

The *respiratory functions* undergo modifications in rate and rhythm in consequence of cerebral disease. In exaltation of the cerebral functions the respirations may be extremely frequent, while the rate may be much lessened by lowering of these processes. The rhythm is often modified in cerebral affections. The respirations may become simply irregular in rhythm as well as depth, or the peculiar type of periodic breathing, already described as "Cheyne-Stokes respiration," may be present. Simple irregularity may occur in exalted or depressed conditions of the cerebral functions, but the "Cheyne-Stokes" rhythm only occurs when there is depression.

The *functions of defæcation and micturition* may be disturbed in lesions of the spinal cord affecting the centres in the lumbar enlargement, the common symptom of their implication being paralysis of the sphincters, leading to *incontinence* of fæces and urine. The incontinence may be unobserved by the patient if the sensory functions are disturbed as well as the motor. But if there is only a removal of the control of the cerebral centres over those in the cord, interfering with the voluntary factor required to call the evacuant processes into action, there may be *retention.*

These functions are most commonly disturbed in cases of severe organic disease, such as myelitis or softening.

The *sexual functions* depend on the integrity of their centre in the lumbar enlargement. If the lower part of the cord is not under the control of the higher centres the sexual reflexes are exalted, and *priapism* is the result. If the lower part of the cord is diseased and the sexual centre is involved, the sexual functions are depressed or abolished.

6.—VASO-MOTOR FUNCTIONS.

Disturbances of vaso-motor functions manifest themselves in a variety of different ways.

Changes in the *temperature* occur in many cases, the departure from the normal being most commonly a rise, but not infrequently a fall. These alterations are local or general, according to the cause.

Modifications in the *colour of the skin* are also frequent —flushing and pallor being seen over larger or smaller regions of the surface.

Alterations in the *amount of perspiration* may also be present in the form of excess or deficiency. These changes may be local or general. It is open to question whether in such symptoms there is not also a disturbance of the secretory nerves.

In addition to these external symptoms of vaso-motor disturbance, there may be others connected with secretory apparatus, in which, however, it is possible that there is, along with the vascular change, some interference with the nerves of secretion.

Excessive or modified secretion from the *lachrymal glands* may be found in certain affections.

Excessive or modified secretion from the *salivary glands* may be observed in some cases.

Changes in the *amount of the urine* are common—*oliguria*, or a deficient flow, being observed, as well as *polyuria*, or an excessive flow.

There may be alterations in the *character of the urine*, the most common of these being *albuminuria*, dependent on some transitory affection of the vessels and epithelium of the kidney, resulting from vaso-motor disturbance, and *glycosuria*, produced by some interference with the vascular supply to the liver, which is usually brought about by affections involving the hepatic vaso-motor centre in the medulla oblongata.

These vaso-motor changes are extremely frequent in cases of hysteria, but, as above mentioned, they are also found as the result of structural lesions.

7.—TROPHIC FUNCTIONS.

Interference with trophic functions is frequent in nervous disease.

Changes in the *nutrition of the skin* may result from functional or organic disease. Various skin rashes, as well as atrophies and hypertrophies of skin appendages, may be found without any definite organic disease of the nervous system, but they are often, on the other hand, associated with them.

Sloughing of the skin is frequent in such grave structural changes as myelitis.

Atrophy of the hair and nails may be observed in some cases where there is some local or general disturbance of the nervous system.

Wasting of the muscles occurring rapidly, as already referred to, shows some disease of the multipolar cells of the anterior cornua of the cord.

Deformities of bones and joints are frequently found in connection with locomotor ataxy.

8.—MENTAL FUNCTIONS.

In considering disturbances of the mental processes we have to pay attention to sleep, consciousness, and speech.

SLEEP.

The states of *sleeping and waking* may be modified to excess in each of these two directions, the tendency to excess of sleep being termed *somnolence*, while the opposite is *insomnia*. Such departures from the normal may be only a slight exaggeration of an individual idiosyncrasy, or may result from nerve-exhaustion, or may mark the presence of some definite lesion : the cause of such changes must be determined by a consideration of all the associated symptoms.

CONSCIOUSNESS.

The condition known as *consciousness*, which we may for clinical purposes assume to be the outward manifestation of perceptive processes, undergoes more or less entire abolition or perversion in many cases. When the patient gives no evidence of perception spontaneously, or in response to external stimuli, he is said to be *unconscious*. There are degrees of unconsciousness : when partial it is termed *stupor*, when profound it is called *coma*. When the patient lies in a state of deep unconsciousness with the eyes open, the condition is termed *coma-vigil*. In stupor the reflexes are not entirely abolished; the iris contracts in response to a bright light, and a painful stimulus will induce movement. In coma, on the other hand, all superficial reflex functions are abolished. These states are frequently accompanied by some of the alterations of the organic reflexes which have already been described, such as changes in the circulation and respiration.

Sometimes, from the symptoms present and their mode of onset, it is easy to diagnose the cause of the unconsciousness; at other times it is extremely difficult. The loss of consciousness may be the result of some organic lesion of the cerebrum, such as hæmorrhage or inflammation; of some explosive affection, such as epilepsy; of some stoppage of the circulation, such as syncope as a general, or embolism as a local cause; or of some toxæmic condition, as in fevers, uræmia, or narcosis. For a full discussion of the means by which these different causes may be diagnosed, special works must be consulted. The diagnosis must turn on the entire group of symptoms, not on one alone, and the mode of invasion is of high importance.

The *perversions of consciousness* presented in disease may be grouped together under the term *delirium*. This term is usually restricted to those symptoms which result from some of the causes which have just been referred to in the preceding paragraph, and it corresponds to the condition known in psychological medicine as *insanity*. Delirium is more common in such conditions as fever and narcosis than in cerebral disease.

There are three well-marked classes of mental conditions common in delirium. The impressions made upon the senses may arouse false sensory perceptions, which are termed *illusions;* or sensory images may arise without any impressions upon the senses, which are termed *hallucinations;* or false ideas may be present, which are termed *delusions*.

SPEECH.

Disturbances of *speech*, using this term in its widest sense, present one of the most difficult problems which have to be dealt with by the physician. A patient may be unable to speak on account of disorders of the mental processes, or from affections of the muscles concerned in articulation. With such alterations of the faculty of conveying ideas, we have nothing to do in this place. We have to confine ourselves to disorders of speech unconnected with mental disturbances, and independent of muscular affections.

In order to grasp the bearings of this subject, attention must be bestowed upon the perception as well as the production of spoken and written language, *i.e.*, the employment of conventional symbols as a basis for the communication of ideas. There are receptive as well as emissive functions connected with language.

Disorders of the faculty of communicating thoughts, arising from defect of the cerebral speech processes, are classed under the term *aphasia*. There are disturbances of the receptive as well as of the emissive functions. The former are grouped in a class called *sensory aphasia*; the latter constitute *motor aphasia*.

Sensory Aphasia.—A patient is sometimes found who has lost the power of understanding printed or written words. He can understand spoken language, and can himself speak perfectly. The eyesight may be practically intact, and the person suffering from this affection may be able to make a copy of any specimen of printed or written words, and yet cannot understand their meaning. He may even be able to write down his thoughts, but after having done so he is unable to read what he has written. This form of sensory aphasia is usually called *word-blindness*, and the situation of the lesion in such cases is found to be in the occipital lobe of the brain—at the spot previously referred to as the centre for the reception of visual impressions.

Cases are also observed in which spoken language cannot be understood. The patients in such cases can read and write. Their hearing for sounds of ordinary kinds may be but slightly interfered with, but is more usually in a great degree lost. They can speak, but on account of their want of comprehension of their own spoken words, they are apt to make many mistakes. This form of sensory aphasia is termed *word-deafness*, and it depends on a lesion of the superior temporo-sphenoidal convolution, where the centre for the reception of auditory impressions is situated.

Motor Aphasia.—Patients suffering from cerebral disease may be able to think perfectly well, and to understand spoken or written language (the latter, however, in many cases being less easily grasped), without being able in any way by speech or writing to express their thoughts. There are several degrees of this condition, depending upon the extent of the lesion and the site which it occupies, and to these different terms have been applied. It is sufficient for the present purpose to group these different varieties together under the name of *motor aphasia.* The position of the lesion in cases of complete motor aphasia is in the posterior part of the third frontal convolution, and perhaps also in the neighbouring part of the ascending convolution.

The lesions in cases of aphasia are most commonly, but not invariably, situated in the left hemisphere. They are, in fact, found in the left hemisphere in right-handed persons; in the opposite hemisphere when the patients are left-handed.

CHAPTER X.

EXAMINATION OF THE EYE, EAR, LARYNX, AND NASO-PHARYNX.

The Eye.

The objective examination of the eye is necessarily limited to those parts which we can handle or see, so that the methods at our service are—

I. Palpation.
II. Inspection.

The former is too often neglected by the physician, whether because, as compared with the latter, it appears to yield little information, or because its performance occupies so little time that it does not impress his mind as a student, we shall not stay to discuss; but we give it the first place here, lest by relegating it to the close of the optical examination we help to perpetuate that neglect through which too many eyes are now sacrificed.

I. Palpation.

The chief application of this method is found in the determination of the state of the intra-ocular tension. The fluid contents of the eyeball are constantly being changed by secretion of aqueous humor from the ciliary processes and back of the iris, and by the escape of this fluid into lymph spaces at the periphery of the anterior chamber, and in slight amount at the optic disc.

In health the inflow and outflow are so balanced that the contents of the globe practically maintain a constant quantity, and, exerting a uniform pressure upon the coats of the eye, offer an elastic sense of resistance to compression. This resistance is called the *tension* of the eye, and it increases or diminishes directly as the contents of the globe. It corresponds to what surgeons term "fluctuation."

To elicit it, direct the patient to look downwards without lowering his head, and to close his eyelids gently.

The observer steadies the globe against the floor of the orbit by lightly resting the pulp of one index finger on the upper lid; with the other he makes gentle intermittent pressure upon the eye, and mentally compares the sense of resistance with that offered by a normal eye.

The patient's two eyes should be contrasted with one another, and if in doubt, with those of a healthy person. In some persons the globes feel much firmer than in others; allowance must also be made for the thickness and softness of the lid.

Following Bowman's suggestion, it is convenient to express the results obtained thus :—

T.n. = tension normal.
T + (?) = ,, increased probably.
T + 1 = ,, ,, certainly.
T + 2 = ,, ,, but globe can be dimpled.
T + 3 = stony hardness.
T − (?) = tension diminished probably.
T − 1 = ,, ,, certainly.
T − 2 = ,, much diminished.
T − 3 = globe flaccid.

Increased tension is intimately related to that morbid condition of the eye known as glaucoma, and is a sign of the gravest import, since it is usually associated with marked deterioration of vision, and is capable of destroying it altogether with great rapidity. The tension undoubtedly changes in some persons at short intervals. In the case of adult persons, with or without external signs of inflammation of the eyes, complaining of failure of vision, and especially of paroxysmal pain, and particularly when mydriatics are being employed, the tension should be carefully examined *at each visit.* For want of this precaution too many eyes are treated for supposed neuralgia or cataract, and the surgeon's aid is not invoked until too late.

New growths within the eye may also cause increased tension.

Diminished tension usually indicates an unnaturally fluid state of the vitreous, following escape or shrinking of that body, and this again is frequently associated with detachment of the retina.

Various mechanical tonometers (*i.e.*, tension measurers) have been devised, but their practical utility is scarcely yet established. Probably the educated sense of touch will always be the most accurate, as it certainly is the most ready, method of tonometry.

Palpation with one or more fingers is also of value in revealing increased or diminished sensitiveness of the globe to pressure, and often throws light upon the seat, nature, and extent of new growths or inflammatory changes affecting the position of the eyeball, or modifying its shape.

In retrobulbar neuritis some sense of pain may often be elicited by pressing the eyeball backwards into the orbit. This may be best done by standing at the side of or behind the patient, steadying the back of his head with one hand, and gently pressing upon the closed lids with the pulps of the first three fingers of the other hand, held with the palm against the patient's cheek. The same means may be employed to determine whether proptosis of the eyeball is due to a solid new growth in the back of the orbit, or some more compressible and less serious tissue alteration, such as vascular engorgement.

II. INSPECTION.

We have now to determine objectively the state of the refractive media—cornea, aqueous, crystalline lens, and vitreous—first as to—

(*a*.) Transparency,
(*b*.) Refractive power;

and secondly, the condition of the coats of the eye viewed from within.

(a.) *Transparency.*

Simple inspection.—For the examination of the *anterior segment* of the eye the patient should be placed facing a window or other good source of light.

The illumination can be heightened by focussing the light upon the eye by means of a lens.

Bright sunlight should of course be avoided, since the heat rays are focussed as well as those of light. If the room be darkened, a bright steady gas, oil, or candle flame may be used, and should be placed at a distance of at least 18 inches from the side of the patient's head, and slightly in front of the plane of his face, so that the light falls obliquely upon it.

Oblique focal illumination.—The observer, steadying his hand by resting the little finger on the patient's cheek or temple, should support a lens of 2 or 3 inches focus between his thumb and forefinger, and with it concentrate the light upon any desired spot. By moving the lens nearer to or further from the eye, the light can be brought to bear on any part of the anterior segment of the globe, as deep as the posterior aspect of the crystalline lens and anterior layers of the vitreous.

Whether employing simple or oblique focal illumination, the observer may aid his sight by holding another lens as a magnifying glass between his own eye and that of the patient. Opacities in the cornea, aqueous humor, and lens should be carefully sought for.

To explore the *posterior segment* of the eye the ophthalmoscope is required.

The Ophthalmoscope.—Every pencil of light which enters the eye through the pupil is bent by the refractive media towards a focal point on or near the retina. In like manner the reflected rays which escape from the interior of the eye are, by the same relation of the media to the outer air, caused to return almost along the directions by which they originally came. It is therefore impossible for the eye of an observer to receive the returning rays without at the same time intercepting those which are passing to the observed eye, or, in common phraseology, "putting his head in the light."

Hence the pupil appears black, or so few rays reach the observer's eye that at the most only a faint reddish glow, and no satisfactory picture of the interior, can be obtained.

By placing an artificial light behind the plane of the patient's face, and using a mirror to reflect rays into his eye, the mirror becomes the apparent source of light, and the returning rays tend to be restored towards it. A small aperture at the centre of the mirror allows some of them to enter an observer's eye if placed immediately behind it.

What will now be seen will depend on the size of the pupil, the transparency of the media, their refractive power, and the distance of the observer from the eye observed.

In a normal eye, at 18 inches distance, a beautiful rosy red glow will appear through the pupil, and is due to the light being reflected from the very vascular retina and choroid. The tint will vary greatly with the complexion of the individual and the amount of pigment present in the hexagonal epithelial layer of the retina and connective tissue stroma of the choroid.

If the pupil be small it may be dilated by instilling a drop of a watery solution of hydrobromate of homatropine (4 grs.-℥i.) twenty minutes before the examination. The mydriatic effect of this substance asserts itself rather more quickly than a corresponding application of atropine, and passes off more rapidly. In many persons a few drops of a solution of cocaïne (four per cent.), applied at intervals of a few minutes, is an efficient mydriatic, and has the advantage of not impairing the power of accommodation.

The patient should now be successively requested to look upwards, downwards, to right and left, and finally straight forward, while the position of his head remains unaltered.

In this way any *fixed* opacity in the vitreous (as well as any corneal nebula or striæ in the lens) will probably be brought into view, especially if the observer take care to examine the peripheral parts by moving his own head in a contrary direction to that taken by the patient's eye.

If the same movements be rapidly repeated by the patient any *free* opacities which have sunk to the bottom of the vitreous will be thrown upwards, and be seen floating or falling as dark dots, threads, or cloudy masses. Brilliantly glittering crystals of cholesterin are sometimes seen. All indicate an unhealthy state of the vitreous.

By placing a lens of suitable strength behind the sight-hole of the mirror and approaching within two inches of the patient's eye, we can examine the vitreous at any plane, scrutinise in detail any fixed opacity, and tell its distance from the retina.

(*b*.) *Refractive Power of the Ocular Media.*

[*Refraction.*—If a ray of light travelling in one medium (*e.g.*, air) enters another of different density (*e.g.*, glass or aqueous humor) obliquely, it is bent from its original direction, or undergoes what is termed "refraction."

The refractive power of a medium depends on its density and the relation of its surface to the adjacent medium. In physiological optics the media with which we have to deal are air, glass, and the transparent tissues of the eyeball; the last two being in the form of lenses.

The strength of a lens is expressed in terms of its refractive power on a standard pencil of light (namely, one composed of parallel rays) travelling in air.

A Dioptre.—In physiological optics *a lens which can bend parallel rays to a focus at a distance of one metre from its centre* is taken as the unit of lens measurement, and is called a lens of *one dioptre or dioptric.* As the meaning of this term is somewhat perplexing, we may explain its origin. In the older works on optics matters relating to the *reflection* of light were discussed under the heading "Catoptrics" (κάτοπτρον = a mirror), while those relating to *refraction* of light were termed "Dioptrics" (διόπτομαι = to look through). Hence the unit of dioptric (refractive) measurement has been called a dioptre.

If a lens has only half the refractive power of the unit lens its focal point will be at twice the distance, namely, 2 metres. If it be twice as strong as the unit lens, the focal point will be at half the distance; if thrice as strong, at a third of the distance, and so on.

Only convex lenses can produce *positive*, *i.e.*, real foci. A concave lens causes divergence of rays, but its refractive power can be expressed in terms of its *negative* focus, *i.e.*, the point from which it makes parallel rays appear to come.

Lenses are accordingly numbered thus :—

Convex.—

+0·25 D.	Focal distance,	4 metres,	*or*	(roughly)	160 inches.
+0·5 D.	,,	2 ,,		,,	80 ,,
+0·75 D.	,,	1·5 ,,		,,	60 ,,
+1 D.	,,	1 metre.		,,	40 ,,
+2 D.	,,	·5 ,, or 50 centim.		,,	20 ,,

And so on.

Concave.—

−0·25 D. −0·5 D. −0·75 D. −1 D. and so on.

These have the same *negative* focal distances as the *convex* lenses of corresponding strength.

Spherical lenses are those whose surfaces are arcs of spheres, and have the same refractive power in every meridian.

Cylindrical lenses are curved only in one direction. Rays which enter such a lens in planes parallel to the axis of the cylinder pass through unchanged. Those in planes at right angles to the axis meet with curved surfaces and undergo refraction. Cylindrical lenses may be convex or concave, and are numbered like sphericals. In writing we indicate the difference thus (say), +1 D sph. or +1 D cyl., −1 D sph. or −1 D cyl.]

The refractive media of the eye form a combined system of lenses whose principal focal distance in a normal eye is 22·231 mm. (equal to a single lens of about + 45 D sph.). The rods and cones of the retina occupy the focal plane.

Under the subjective examination (see p. 301) we have already described the refractive errors which may be met with, and their correction by subjective means.

There are various objective methods of greater or less accuracy whereby they may also be detected, such as the shadow test (retinoscopy); the apparent direction of movement of the optic disc or blood-vessels when seen with the ophthalmoscopic mirror alone at some distance from the patient's eye; the lens required to give the clearest image by the direct method of ophthalmoscopy; and the alteration of the size of the optic disc when the object lens is withdrawn or advanced slightly in the indirect method of examination.

The Shadow Test (*Retinoscopy*, *Pupilloscopy*, *Keratoscopy*, or the *Flash Test* are identical terms).

If a beam of light be thrown with an ophthalmoscopic mirror into a healthy eye *from a distance of* 4 *feet*, it will be more or less accurately focussed on the retina, and the reddish glow seen through the pupil will be brightest when the eye is emmetropic, and less bright in proportion as the eye is hypermetropic or myopic.

If the mirror now be tilted so as to make the light move over the eye from side to side, or up and down, the spot of light on the retina will also move, and with it that area of shadow which always surrounds focussed light.

The following phenomena may be observed, and what is true of the glow is true of the shadow.

(1.) With a *concave* mirror of 8 or 9 inches focus the retinal glow moves in the *opposite* direction to (technically called "*against*") the mirror in emmetropia, hypermetropia, and in myopia of less than 1 dioptre.

If the myopia exceed 1 dioptre the retinal glow appears to move in the *same* direction as the mirror is tilted (technically called "*with*" the mirror).

(2.) With a *plane* mirror in emmetropia, hypermetropia, and low myopia the glow moves "*with*," in higher myopia it appears to move "*against*" the mirror. It is then essential for the observer to note first the kind of mirror he is employing.

The *brilliance* of the glow and the *direction* of its movement are alone sufficient to indicate the presence, nature, and to some extent the degree of a refractive error; but the amount of error may be ascertained with very great accuracy thus:—

By successively placing convex lenses of increasing strength close up to and immediately in front of an eye which we have determined by the shadow test to be emmetropic, hypermetropic, or feebly myopic, we can alter the direction of the rays as they leave the patient's eye and make them appear to come from an eye which has 1 dioptre of myopia.

If this effect is produced by a lens of less than + 1 D sph., the observed eye is *feebly myopic.*

If + 1 D sph. is required the eye is *emmetropic.*

If a stronger convex glass is needed the eye is *hypermetropic.* From the *weakest* convex lens which makes the movement of the glow appear to be myopic, *subtract* 1 *dioptre;* the remainder expresses the amount of hypermetropia present.

On the other hand, if the original movement reveals myopia, concave lenses of increasing strength should be employed. The *weakest* which *reverses the direction of movement of the glow* is taken as the measure of the *myopia.*

Since any exercise of accommodation by the patient will increase the refractive power of his media, he should be directed to gaze at a distant object at the lowest possible level over the observer's head, in order that the test may be applied to the macular region.

For absolute accuracy a mydriatic should be employed.

The pupil is then large, the patient can look straight at the mirror and cannot accommodate.

This test is of the greatest value for the detection of astigmatism, since the movement in any meridian shows the refractive state of that meridian.

When the ocular refraction is not absolutely spherical (and it seldom is), the meridians of greatest and least curvature are either respectively vertical and horizontal, or may be oblique, but always at right angles to one another (except in irregular astigmatism).

While investigating the refraction as above explained, the observer's attention should be concentrated first on the correction of that meridian in which the movement of the glow is most easily seen, and then on that at right angles to it. The difference between the correcting glasses required is due to astigmatism, and is the measure of it.

The first meridian may be corrected with a spherical lens, and the second more conveniently with a cylinder.

The apparent movement of Objects on the Fundus.

If with an ophthalmoscopic mirror *alone* an observer seated *at a distance of* 30 *inches* from a patient perceives not merely a rosy glow, but a distinct image of part of a blood-vessel on the optic disc, he should move his head from side to side, keeping the light on the patient's eye and the mirror before his own.

If now the retinal object appears to move in the *same* direction as the observer's head, the eye is *hypermetropic;* but if in the *opposite* direction, there is myopia of at least 2 dioptres present, and may be much more.

THE EXAMINATION OF THE FUNDUS OCULI.

There are two methods whereby we may view the inner surface of the globe.

(1.) *The direct method* is that in which the observer, approaching with his ophthalmoscope to within 1 or 2 inches, reflects the light into the patient's eye, and obtains an erect image of a small portion of the fundus enlarged about twenty times (in emmetropia). In this case the patient's media act as a simple magnifying glass.

To get the full advantage of this method, the observer should employ a compound ophthalmoscope, and must not exert his accommodation. If he has himself any refractive error, he must commence the examination with that lens behind the aperture of his mirror which corrects his vision for distant objects (*i.e.*, for parallel rays).

If the patient's eye be emmetropic, the fundus will now be readily seen, since the rays emerging from an emmetropic eye are disposed in parallel pencils. But if the patient be hypermetropic, a convex lens, if myopic a concave lens, will be required to render the rays parallel. This lens will give the most distinct image, and provided the observation be made in relation to the patient's macula, *will be at the same time the measure of his refractive error.*

If the observer be emmetropic, he will readily select the required lens, if any, by bringing plus or minus lenses of increasing strength behind the mirror aperture. If himself ametropic, and therefore employing a lens to correct his own refraction, he must increase or diminish its strength until the clearest image is obtained. The difference between the lens with which he commenced and that with which he concluded is the measure in dioptres of the patient's error.

The source of illumination should be placed behind and to the same side of the patient's head as the eye which is to be examined. The observer should use his right eye to examine the right eye of the patient, and his left eye for the left.

(2.) *The indirect method* is that in which the observer, holding the mirror in one hand before his eye, reflects light into the eye of the patient from a distance not less than 18 inches. In his other hand he supports a convex lens of 3 inches focus at its focal distance from the patient's eye, resting his little finger on the patient's brow or cheek. By means of this lens the beam of light reflected from the mirror is not only concentrated so that nearly the whole of it enters the patient's pupil, but the returning rays reflected from the fundus, after emerging from the eye, are bent to foci in the air, and form an inverted image of a portion of the fundus (enlarged in emmetropia about five times) at or about the focal distance of the lens on the side nearest to the observer. This image, being situated 10 or 12 inches in front of the observer's eye, will be seen by such an effort of accommodation, or the use of such a lens behind the mirror aperture, as is required by the observer in order to read small print at that distance.

The source of illumination should be so placed that the patient's face is in shadow, and the hand holding the lens does not intercept the light on its way to the mirror. The observer should hold the mirror in his right hand when using his right eye, and in his left when using his left eye.

A definite method of conducting the exploration of the fundus is essential, and an examination of parts in the following order is recommended.

The optic disc should be first sought for. The optic nerve pierces the globe about 3·5 millimetres to the inner side of the posterior pole. To bring the disc into the observer's line of view the patient must turn his eye slightly inwards. For one eye the tip of the extended little finger of the hand holding the mirror, for the other the ear on the opposite side of the observer's head, gives a convenient indication of the direction in which the examined eye must be turned.

The apparent *size and shape* of the disc depend chiefly on the refractive condition under which it is viewed.

The normal shape is circular or slightly oval, with the long axis vertical.

In hypermetropia it appears small by the indirect method; in myopia large, and as the lens is withdrawn it becomes *smaller* in the former case; *larger* in the latter. Consequently in astigmatism the disc may appear decidedly oval, the more so as the lens is withdrawn, and the shortest axis corresponds to the meridian of least, the longest to that of highest refractive power.

The normal *colour* of the disc is roseate from its capillary blood supply, but it is considerably paler in tint than the surrounding fundus.

Its *edge* is usually well defined, and often exhibits a fine white circle—" sclerotic ring "—bounded by a dark border—" choroidal ring "—the free edge of the choroid.

The *surface* of the disc usually presents a physiological pit at or near the centre.

It is the whitest part of the disc, being nearest the sclerotic, and is produced by the centrifugal disposition of the nerve fibres as they expand on to the retina.

The nerve cylinders usually lose their white sheaths before piercing the lamina cribrosa. Occasionally some persist for a variable distance beyond the disc margin, producing pure white patches with striated edges which radiate from the disc. This innocent congenital condition is termed *opaque nerve fibres*, and is only of importance when mistaken for some serious lesion.

The macula will be found two discs' breadths to the *real* outer side of the disc. It should be seen when the patient looks directly towards the mirror. It is an area devoid of large blood-vessels, sometimes indicated by a deeper pigmentation of the fundus, frequently bounded by an oval silvery halo of light reflected from its shelving margin, and often presenting a bright yellowish point at its centre—the fovea centralis.

The retinal vessels generally pierce the disc within the " pit." The arteria centralis retinæ, before or immediately after its emergence, bifurcates into an ascending and descending trunk, each of these again divides on the disc into one or more main vessels, which pursue sinuous arched courses inwards or outwards, giving off numerous branches to supply the retina generally. Corresponding veins converge to the disc. The main arteries are paler in tint, and have about two-thirds the diameter of the veins.

Pulsation of one or more of the veins on the disc is of frequent occurrence.

Pulsation of the arteries has been seen in glaucoma and aortic incompetence, and may be induced by artificial pressure on the globe.

By following the retinal vessels in their distribution, the general surface of the retina can be explored with the greatest accuracy and completeness. It must never be forgotten that the image obtained by the indirect method is inverted and reversed.

Some of the more important morbid appearances of the *disc* and *retina* may be briefly described.

Anœmia may accompany profound general anæmia, and small linear or irregular hæmorrhages are sometimes found in severe and pernicious cases.

Hyperœmia of the disc produces an approximation of its colour to that of the fundus, and makes its margin less defined. Not infrequent in hypermetropia.

Inflammation of the optic nerve can only be diagnosed ophthalmoscopically when the optic disc is involved. The general term for the former is *optic neuritis ;* for the latter *papillitis* is a better term.

Papillitis may occur in one or both eyes independently of general affection of the retina—*retinitis,* or in association with it—*neuro-retinitis,* or in slighter degree may participate in *choroiditis.*

Signs.—Increased redness or fluffy reddish-greyness, blurring of the edges by exudation, tumefaction, expansion, and striation of the surface, distention of retinal veins, narrowing of arteries, and obscuration of many of these vessels on the disc.

Its presence in both eyes affords a most important confirmation of the diagnosis, when other symptoms point to cerebral tumour; but among other causes are syphilis, lead poisoning, and cold.

Owing to the natural vascularity of the part, *hyperæmia of the retina* cannot be safely diagnosed until tortuosity and distention of the veins, with commencing cloudiness of the membrane from exudation, especially along the lines of the vessels and at the disc margin, indicate that *retinitis* is actually present.

Moderate congestion, with a steamy appearance of the fundus, characterises *serous retinitis* (some believe it to be due to *choroiditis*), and is probably syphilitic, especially if dust-like opacities occupy the vitreous humor. More marked congestion, with flame-like or linear hæmorrhages scattered over the fundus, may justify the term *hæmorrhagic retinitis*.

In *albuminuric retinitis* the intensity of the signs varies greatly, and these are not characteristic until the fogginess of the retina about the disc and macula shows signs of degeneration in the form of glistening white dots radiating in rows from the fovea centralis. Some of these frequently coalesce into patches of various shape and size. Small spots of hæmorrhage are frequent. In any case an absolute diagnosis should not be given until the urine has been examined, since similar appearances are sometimes found in diabetes and other less understood conditions. Inflammation of the disc and retina may resolve or pass into atrophy.

In *optic atrophy* there is pallor of the disc from fibrosis, and loss of capillary blood supply. If the retina is involved, there is also shrinking of the retinal vessels, and frequently white lines along their edges from fibrosis of their walls.

Mere pallor of the disc does not imply atrophy unless confirmed by subjective examination.

Embolism of the central artery causes sudden blindness. In thrombosis the onset is less sudden. In both, marked pallor of the fundus, great constriction of arteries, narrowing of the veins on the disc, with some increase in size towards the periphery, are the earliest signs: within forty-eight hours foggy œdema of the retina about the disc, and especially at the macula, with a plum-coloured spot at the fovea. Absorption of the œdema and atrophy results in a few weeks. If only one branch is plugged, the appearances are limited to the area supplied. Cardiac or arterial disease should be looked for.

Retinitis pigmentosa is a slowly progressive non-inflammatory degeneration of the retina characterised by the deposition of pigment in long lines upon the coats of the retinal vessels, or in branching spider-like spots, forming together a lace-like pattern disposed in a zone intermediate between the equator and posterior pole of the eye. Shrinking of the vessels, and hazy waxy pallor of the disc, are further indications of the atrophic process.

Detachment of the retina may be traumatic or idiopathic.

If recent, slight, and transparent, the diagnosis must depend on local darkening and tortuosity of vessels and relatively hypermetropic refraction as compared with the rest of the fundus on examination by the direct method. By the indirect method, if the lens be moved from side to side, a detached portion of the retina (being focussed nearer to the observer) appears to move more freely than the attached membrane,—just as when one eye is closed and two objects in the same line are viewed the nearer appears to move over the more distant when the observer sways from side to side. This is termed "parallactic movement." If the detached retina has any folds or rucks, as is commonly the case when atrophied or extensive, silvery grey reflections of light occur from their surfaces, and may be readily detected with the mirror alone at a distance of 8 or 10 inches. A floating billowy grey membrane, with dark bloodless vessels coursing over the crests, is typical. If it does not quiver as the eye is moved, an intra-ocular tumour should be suspected.

THE CHOROID is only visible in proportion as the hexagonal pigment layer of the retina is deficient, and then presents numerous dull or brick-red vessels disposed in leashes, or a network with pigmented connective tissue between.

Acute choroiditis is commonly accompanied by exudation into the vitreous, which further screens the process.

The commoner appearances are the results of previous disease which have led to thinning or atrophy of the membrane and pigmentation. A crescentic defect in the choroid at one (usually the outer) side of the disc is so frequent in myopia as to be called a *myopic crescent*. When larger, it may surround the whole disc and extend irregularly—*staphyloma posticum*. Its colour is white, because the sclerotic forms the back-ground. It may be pigmented, especially at its margin. The retinal vessels pass distinctly superficial to it. The macula is a frequent seat of choroidal atrophy and pigmentation—*central choroiditis*. There is a senile form, which presents numerous small yellowish-white spots, which tend to coalesce, and may undergo atrophy and pigmentation. This should be looked for when sight fails in old persons.

In *disseminated choroiditis* white patches of atrophy, with or without pigment, varying in number and size, are scattered irregularly over the fundus. When small and well-defined, enclosing a spot or bounded by a ring of pigment, syphilis is strongly suggested.

Hæmorrhages and pigmentation of the choroid tend to assume coarser proportions than in the retina, and are often crossed by the retinal vessels.

Tubercles in the choroid are rare, but may occur in general tuberculosis, usually at a late stage. They usually present isolated prominent yellowish rounded spots situated about the posterior pole of the eye, behind the plane of the retinal vessels, and free from atrophy or pigmentation.

The term GLAUCOMA is applied to a group of affections accompanied by increased intra-ocular tension (see p. 342).

In *acute glaucoma* there is usually a dull or steamy condition of the media, which may entirely obscure the state of the fundus. In severe cases retinal hæmorrhages may be visible, or extensive hæmorrhage may complete the shroud.

Pulsation in the retinal *arteries* on the disc is not uncommon.

Complete cupping or retrocession of the whole surface of the disc from long-continued pressure is characteristic of the more *chronic* forms of the disease. A bluish pallor within the cup, and a yellowish halo around its margin, indicate the atrophy by which it is usually accompanied. Abrupt turning of the vessels over the edge of the disc, apparent loss of continuity in their course, and parallactic movement of the edge of the disc in relation to its floor, are further proofs of deep cupping. These, however, are but the final appearances of this insidious malady. The state of the intra-ocular tension and the subjective symptoms are the more essential factors in the diagnosis of the disease at the time when it is remediable.

Examination of the Ear.

The inspection of the meatus and tympanic membrane is best done by using a reflector (focal distance about 5 inches) and a suitable ear speculum. The best light for the purpose is got from a window on a tolerably clear day; a candle will, however, be found sufficient. The patient is seated with the ear to be examined away from the light. A speculum is then introduced into the meatus up to the commencement of its osseous portion, the auricle being at the same time pulled upwards and backwards in order to straighten the canal. Any obstruction in the latter, if present, will in this way be seen; otherwise a view of the membrane tympani is obtained. This structure in the normal condition shows—(1.) The handle of the malleus running from its anterior and upper part, downwards and backwards to a little below the centre as a yellow unmistakeably osseous ridge; (2.) Meeting this at an obtuse angle (open anteriorly) is seen the bright spot, usually triangular in shape and always conspicuous by its lustre; (3.) The membrane itself, oval in shape, and bluish grey in colour; (4.) The upper part of the handle of the malleus called the short process, from which run two delicate folds

—the more marked passing backwards, and the other forwards (the posterior and anterior folds.)

It would be useless here to discuss the various deviations from the normal met with; those who desire information on these points we must refer to works on otology.

Having obtained a view of the drum membrane, it may be desirable to test its mobility. This can be done by directing the patient to hold the nose and blow. If the Eustachian tubes be pervious the membrane can be seen to move if inspected during this act. If this experiment (known as Valsalva's) be impossible, then air must be driven through the Eustachian tube by means of Politzer's method (or one of its modifications), or the Eustachian catheter, by an assistant while the ear is inspected; if no assistant be at hand, a pneumatic or suction speculum (known as Siegle's) may be employed.

In order to determine the condition of the Eustachian tube it is desirable to drive air from the nose into the middle ear, while the ear of the observer is connected with that of the patient by means of an auscultating tube. For therapeutic purposes this may be accomplished by Politzer's method, which consists in introducing the perforated nozzle of an indiarubber bag into one nostril, while so much of it as is not filled, together with the opposite nostril, are compressed with the finger and thumb. The patient is now directed to swallow a little water which has been previously taken into the mouth. At the moment of swallowing the bag is suddenly compressed, and air is felt by the patient and heard by the operator to inpinge upon the drum membrane. Instead of swallowing, the patient may be directed to say "huck," blow out the cheeks, &c. Fortunately the act of crying also serves the same purpose.

If, however, it be desired to appreciate fine differences in the sounds produced by air passing through the Eustachian tube, it is better to pass the Eustachian catheter. This is a curved instrument having at its outer extremity a wide orifice, into which the nozzle of an air-bag fits. At this end there is also a ring corresponding to the curve on the beak. The best method of introducing the catheter is to pass it along the floor of the inferior meatus of the nose until it reaches the posterior wall of the pharynx; the beak is then turned inwards and the instrument withdrawn until the curve hooks upon the septum of the nose. If the catheter be now rotated until the ring (which corresponds to the point) is directed towards the outer canthus, the instrument will, as a rule, enter the Eustachian tube. It must here be mentioned that difficulties are often met with in catheterisation of the Eustachian tubes, and then other methods must be employed, such as will be found described in any work on diseases of the ear.

After the catheter is *in situ*, and while the ear of the observer is connected with that of the patient, air is driven through the instrument by means of an indiarubber air-bag. The following auscultatory phenomena are capable of detection by a trained ear:—

(1.) A full sound of air impinging against the drum membrane.

(2.) A moist sound far away from the ear, if there be mucus in the pharyngeal orifice of the Eustachian tube.

(3.) A faint moist sound associated with the impact of the air against the drum membrane in some cases in which the tympanum contains fluid.

(4.) A dry whistling sound in narrowing of the Eustachian tube.

(5.) Air whistling through a perforated drum membrane.

Beyond mentioning that bougies are sometimes passed through the Eustachian catheter into the Eustachian tube, we shall not discuss this method of exploration, which should be rarely used, and never except by a skilled aurist.

EXAMINATION OF THE LARYNX.

For examination of the larynx there are required (1) a laryngeal mirror; (2) a powerful light.

It is unnecessary to describe the laryngeal mirror, which is now so well known. It is enough to say that several sizes should always be at the physician's command, and further, that it is, for obvious reasons, well to use one as large as can be tolerated by the patient.

As to light, it is usually well to employ a powerful fixed light, the rays of which are concentrated on the mirror by means of a reflector. The last named may be attached either to a forehead band, or kept in position by a spectacle frame. It should have a central aperture, which, when the mirror is adjusted, should be opposite one of the observer's eyes, so that he can look through it. The actual source of light may be a strong argand burner, a Welsbach or albo-carbon light, or a powerful lamp. The electric and oxy-hydrogen lights will probably for some time to come be only at the command of specialists. The general practitioner, however, has often the best source of light at his command, viz., the sun. As most reflectors are concave, it must be borne in mind that an exact focussing of the rays should be avoided ; otherwise the excessive heat produced will cause the patient pain.

The patient is seated so that the light falls over his shoulder (which side is immaterial) on about a level with his ear. The physician sits opposite him, and so adjusts the reflector that a strong light is directed upon the mouth, which must now be opened. The tongue is then protruded, and held either by the observer's left hand or the patient. To facilitate this, and prevent the fingers from slipping, a pocket-handkerchief is employed, which, together with the hand, must be kept below the lower lip, to avoid shutting off light. The patient is now directed to breathe quietly, and the mirror—previously slightly warmed over the flame—is introduced so that it lifts upon its posterior surface the uvula. The handle of the mirror should be held like a pen, and until the uvula is reached the reflecting surface points downwards ; it is then, however, held more vertically, and the angle which it forms with the horizon is gradually increased until the larynx is brought into view. During these manœuvres it is well to make the patient incline his head slightly backwards. The first part of the larynx seen is usually the epiglottis, which appears in the anterior or upper part of the mirror, while in its lower portion the arytenoid cartilages (surmounted by the cartilages of Sartorini) become visible. If now these last named be followed forwards (*i.e.*, upwards in the mirror) there is seen on each side another little prominence (the cartilage of Wrisberg). Finally, the elevated fold of mucous membrane (the ary-epiglottic fold) runs up to the epiglottis. Immediately inside of this appear two other folds of mucous membrane (the false cords or ventricular bands). If the patient be now told to say "eh," the epiglottis is thrown up, and the false cords come more plainly into view, while the true vocal cords are seen as two white and glistening bands, separated from the ventricular folds on each side by a recess (the sinus of Morgagni). On inspiration the cords are seen to separate, while on expiration they tend to approximate. On phonation they are perceived to be in actual contact. There is one portion of the larynx which can only be properly studied on full inspiration, viz., the fold of mucous membrane between the arytenoid cartilages (the inter-arytenoid fold or posterior commissure). The anterior commissure is best seen, in most persons, just before phonation; here the vocal cords meet, and just above them is a small prominence (the cushion of the epiglottis). During phonation, too, is seen on each side the sinus pyriformis, which lies between the ary-epiglottic folds and the outer wall of the pharynx. The upper surface of the epiglottis should also be examined. It shows a central ligament (the middle glosso-epiglottic) and on each side a fossa (vallecula).

In examining the larynx it is necessary to consider deviations from the normal as to (1) colour, (2) quantity of secretion, (3) swelling or hypertrophy, (4) the presence of tumours (or foreign bodies), (5) localised infiltrations, (6) ulcerations, (7) mobility.

To discuss each of these points would necessitate more space than is at our command. The last mentioned, however, requires a few words of explanation. On normal inspiration the vocal cords are separated, on normal expiration they are approximated, and on phonation they are in contact. It is therefore necessary in each case to study the movements of the cords outwards on inspiration, and inwards on phonation. It must be remembered that it is—in cases of paralysis due to involvement of the recurrent nerve—common for the affected cord to remain immobile in the position of phonation, so that the paresis is only detected on inspiration.

Certain obstacles are often met with in attempting to make a laryngoscopic examination :—

(1.) The patient, owing to nervousness, may hold the breath. This difficulty is usually easily overcome by directing that respiration should go on as usual, and pointing out that it will not be interfered with by the introduction of the mirror.

(2.) Abnormalities of the tongue. In some persons the dorsum linguæ arches itself up so as to leave little or no space between the tongue and the palate. In a few of these cases a tongue depressor has to be employed in order to obtain room for the introduction of the mirror. Occasionally the frenum is so short as to prevent protrusion beyond the lower incisors. Where this is the case our only course is to employ a tongue spatula, and then introduce the mirror in the ordinary way.

(3.) Hyperæsthesia of the pharynx may be so marked as to make laryngoscopic examination well-nigh impossible. This obstacle can almost invariably be overcome by painting the pharynx, and base of the tongue with a solution of cocaïne (10 to 20 per cent.).

(4.) The epiglottis in some persons has a tendency to overhang the larynx and obscure the image. The intonation of "e" may cause it to rise, or, failing this, several rapid short respirations. In exceptional cases these means fail, and it is then necessary to spray the throat with a solution of cocaïne, and afterwards, holding the mirror in the left hand and making the patient hold his tongue, to elevate the epiglottis by means of a laryngeal probe introduced with the right hand. This proceeding, however, requires for its satisfactory execution considerable practice in laryngoscopic manipulation. External palpation of the larynx is sometimes of use—especially in cases of displacement or suspected fracture.

Internal palpation by means of the finger is necessary, particularly in young children, in whom laryngoscopy is often impossible, when the presence of a foreign body is suspected. This method—which must, of course, be very rapidly executed—may also prove of assistance in the diagnosis of œdema glottidis, when the epiglottis and ary-epiglottic folds may be felt as tense prominences.

Sounding or probing the larynx with a bent laryngeal probe directed by means of the laryngoscope is often very valuable. The tongue being held by the patient, the mirror is introduced with the physician's left hand, and the probe, which is bent at a suitable angle, with the right. This method is serviceable in detecting the consistence and attachment of tumours, and also in diagnosing the presence of necrosis. Auscultation in the ordinary sense of the term is not often practised as an aid to laryngoscopic diagnosis. Any interference with the voice should, however, indicate the propriety of a laryngoscopic examination. It must also be borne in mind, however, that paresis of one vocal cord may exist without any interference with phonation.

Dyspnœa due to laryngeal disease is commonly chiefly inspiratory, but in subglottic tumours it may, on the other hand, be expiratory.

Laryngeal cough, especially in children, is characterised in many cases by a peculiar metallic dulness, often spoken of as "croupy."

In doubtful ulcers of the larynx it is often well to remove some of the secretion from their surface by means of a brush or pledget of cotton wool (attached firmly to a suitably bent forceps), and then to examine the secretion so removed for tubercle bacilli.

THE NOSE AND NASO-PHARYNX.

In examining the nose and naso-pharynx we trust as much as possible to sight.

Anterior Rhinoscopy.—This method of examination is carried out with the aid of a strong light and a nasal speculum. In order to concentrate the light upon the nose a laryngeal reflector is employed. The patient is seated as in laryngoscopy, facing the physician. A speculum is then introduced, and that known as the Duplay-Charière is probably the most efficient. When it is used, however, the nose should again be inspected by simply tilting up the tip, in order to exclude a perforation of the anterior part of the septum, which may be covered by one of the blades. The speculum, which consists of two valves separable by means of a screw, is gently introduced straight backwards and then dilated. By this means the inferior turbinated body is seen extending as a thick fleshy mass from the outer wall of the nose towards the septum. In not a few persons this structure, which is composed in great part of erectile tissue, is so large that it obscures everything else, and is in actual contact with the septum. This enlargement may be due to hypertrophy or to simple erection. If the latter only be present then the application of a solution of cocaïne (10 per cent.) will reduce the structure to its normal dimensions. The anterior, and the free margin for a considerable distance backwards, may then be inspected. If now the patient be directed to turn so that the speculum is directed inwards, a considerable portion of the septum can be overlooked and examined. If it be desired to inspect the floor of the inferior meatus, the patient is directed to bend the head forwards; while, in order to see the middle distended body the head is thrown backwards.

The last-named structure then appears as a shelf, extending from the outer wall almost to the septum. Between the two there appears, however, in health a narrow orifice—the olfactory cleft. In order to determine the consistence or point of attachment of any tumour, the presence of carious bone, &c., a probe, guided by the eye, is employed.

Posterior Rhinoscopy.—When it is desired to *examine the posterior nares*, we require a reflector, a small laryngeal mirror, the angle of which, with the shaft, should be somewhat smaller than in the form commonly employed for laryngoscopy, and a tongue depressor. The patient being seated by the side of a strong light, the tongue is depressed, the mirror warmed, and introduced while light is concentrated on the throat. The mirror must be so placed that its reflecting surface is not obscured by the uvula, and points upwards and forwards. If there be ample space between the palate and posterior wall, posterior rhinoscopy is not very difficult, while in some persons it is, owing to insufficient space, impossible. In such cases Voltolini's palate book will be found useful. In others the palate tends to rise too much; the patient must then be directed to breathe through the nose, or else to intone "ng" (or the French word "on"). If the parts be extremely irritable, cocaïne may be applied, as in laryngoscopy. When the mirror has been introduced the first object which usually catches the eye is the septum of the nose (or vomer). To each side of this can be seen the middle turbinated bodies, and below them the upper part of the inferior turbinateds in their posterior aspect come into view. The septum appears as a pale ridge widening at its upper part, while the turbinated bodies are tumour-like and of a more bluish-grey tint. If now the mirror be turned towards the side so that its reflecting surface points upwards and outwards, the orifice of one Eustachian tube is seen as a dimple bounded behind and above by an elevated ridge, posterior to which lies a depression known as the fossa of Rosenmüller. In order to see the vault of the naso-pharynx the reflecting surface must be directed upwards as much as possible. Rhinoscopy, applied to the posterior nares, is by no means an easy manœuvre, and an idea of the whole naso-pharynx and posterior nares can only be got by piecing together the fragments visible in the small mirror employed. Various appliances are employed for holding forward the soft palate so as to obtain more space, but these methods can only be of use to those who have had considerable practice and experience.

It has also been proposed to examine the posterior nares by means of a long speculum introduced through the anterior nares. This method of examination is, however, rarely practised.

In many cases, especially when rhinoscopy is unsuccessful, it is well to explore the posterior nares with the index finger. The patient being seated, the observer, standing at his patient's right side, steadies the head by passing his left arm behind it. The index finger of the right hand is then introduced into the mouth, slipped behind the palate, and the posterior nares rapidly explored. To describe the feeling of the various parts would serve no good purpose; practice alone will enable the physician to derive information from this method of examination. Care must, however, be taken not to mistake the prominent orifices of the Eustachian tubes for tumours. Their occurrence on both sides, and their cartilaginous consistence, will help to prevent such an error.

When examining children it is well to push the cheek between the teeth with the left hand in order to avoid being bitten.

Other objective methods of examination are subordinate to sight and touch applied directly to the part; but in certain cases they are of importance as indicating the necessity for an examination of the nose. Thus a nasal character of the voice, a constantly open mouth, and hanging lower jaw, heavy breathing through the nose, snoring at night, and an offensive discharge from one or both nostrils, are symptoms which are often prominent in nasal disease.

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